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ORIGINAL COMMUNICATIONS.

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SYNDROME-COMPLEX, MENIERE.*

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In considering this lesion as a pathological entity, it must be remembered that many conditions which we now know to be capable of inducing identically the same clinical phenomena were practically unrecognized until a comparatively recent date. It is probable, therefore, that many cases have in the past been recorded as true examples of Ménière's disease which have in reality been the result of other conditions, the nature of which would now be easily recognized. The disease is still more or less wrapped in mystery, which immediate investigations will, I think, serve to clear up. There is hardly a week that passes that I am not consulted in regards to seeing a case, already diagnosed as Ménière's disease. The awe with which the referring physician speaks shows an alarm which is again reflected in the patient, the latter at once feeling that he or she has some severe malady, the like of which was never before experienced.

Historically, Dr. M. P. Ménière, agrege de la Faculte, medecin de l'institution imperiale des Sourds-Muets, cites the following two cases in the *Gazette Medicale de Paris*, 1861, under the title: "Maladies de L'Oreille Interne Offrant les Symptoms de la Congestion Cerebrale Apoplectiforme".

M. X., a doctor, age 47 years, noticed noises of variable nature in his ears for about 15 years, especially in the left, in which the hearing was gradually lost, in spite of the most energetic treatment. The

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hearing on the right was likewise enfeebled, although neither had been at any time inflamed or even the site of a catarrhal lesion. Air had always freely penetrated the middle ear. He often showed symptoms of congestion of the auricles, but only externally. They would become red and warm. The patient alleviated this by simple means, and since 1854 he had given up treatment, hoping that the deafness would remain stationary.

For about three years the disease made no progress until Dec. 26 last, when, after spending a day in the open, M. X., while reading was seized with a fit of violent sneezing. On attempting to stand up he discovered that his gait was unsteady and that he could not walk in a straight line. Three hours later the symptoms persisted, though the patient had taken a foot bath, strong with mustard. He took a light meal and went to bed, hoping that sleep would end the disturbance. He awoke at two in the morning, and found walking still more difficult. He had a sense of weight and pressure in the left occipito-mastoid region, and involuntarily turned from right to left. He had fear of falling, as though the left side of the body did not obey his will, while in bed both sides of the body executed freely all voluntary movements. The intelligence was intact, and the patient was able to write at once to one of his colleagues to ask assistance. He came immediately, and while he was about to bleed the arm, the patient was seized with nausea and vomited. The vomiting was repeated often on the following day, and the stomach would retain absolutely nothing. The 28th, 29th and the following days, under the influence of bleeding, wet cupping and a purgative, the difficulty in movement was progressively dissipated, and on Jan. 2 the patient was able to walk, though still with a little uncertainty of gait. Today the general health is perfect, but the hearing has gradually become poorer.

The physician who had observed on himself exactly the succession of phenomena described here thought at first of a cerebral congestion, and two of his colleagues who had seen him during the illness thought with him that the congestion took place in the cerebellum, but it seems to them today more probable that the attacks depend on a lesion of the internal ear.

The second case: M. X., physician, age 45 years, thin, dark, of nervous constitution and the bilious temperament of Southerners, experienced an attack of intermittent fever in 1858, so severe that he had to use quinin sulphate in large doses. The fever receded, but a ringing in the ears persisted, so severe as to finally draw the attention of the patient. He was less concerned with the sequela, the fever having several recurrences, as under analogous circumstances

the sounds had promptly disappeared. This time it could not be the same as the sounds persisted as soon as it could be determined that the hearing was diminished. There were some changes in the skin of the external meatus—pruritus and a little exudate—but soon the patient experienced vertigo of sudden onset, followed by vomiting. This sort of cerebral disturbance recurred often after a trip or a sudden variation in temperature. The vertigo, with nausea and vomiting, was accompanied by muscular weakness and general depression. During the months of January and February, 1859, the patient was confined to bed. The vertigo disappeared during March and until Aug. 25, when it reappeared with such severity that several times M. X. fell suddenly in the street while going to the hospital. The deafness increased rapidly, and with the exception of a slight mucus exudate the ears were healthy. A slight expiratory effort with the nose and mouth closed was sufficient to introduce air into the tympanic cavity.

All the resources of the most vigorous therapeutics were used on this case, without success and today the hearing is almost gone. The general health is excellent, the cerebral functions are regular and everything goes to prove that the cause of the phenomena observed is to be found in the internal auditory apparatus.

A little later in the same year Ménière writes again the following observations: *Observation 1:* "Forty-two days had elapsed since the appearance of the first cerebral symptoms of which I spoke, when, on Feb. 6, having risen in good health—although my head felt a little heavy—I went to see a patient, my cousin. While I changed the dressing of a fracture I was seized with vertigo, but I was nevertheless able to finish the dressing. I nearly fell, and was given a chair. I experienced eructation and an indescribable illness that the open air did not dispel. I vomited a little coffee, and after an hour of extreme malaise I regained my rooms with much difficulty, leaning on a cane and a stout arm. My gait was tottering and I noted convulsive movements of my face. The left side exhibited strange feelings and then became paralyzed, though incompletely. The disturbance lasted several days. The pulse was neither full nor rapid and the face was not flushed. There was some vertigo and vomiting for two days, and then all the symptoms disappeared, but it was found that my hearing was worse than before the attack."

This time the patient intentionally refrained from bleeding, but took small doses of aloes and calomel to stir up the intestines, and his health promptly returned.

Observation 2: A manufacturer, of X, still young, tall, robust and in the best of health, was giving orders to one of his employees. He

was standing in his office, his arms extended, when his employee saw him suddenly stagger and fall to the floor, where he lay motionless, as though he had been struck by lightning. He was lifted, all his muscles being completely relaxed. His face was pale and bathed in perspiration. Soon nausea appeared, and then vomiting. Consciousness, lost for a moment, returned, and the patient remarked that everything turned about him, this movement making him sick. He noticed at the same time a great noise in his ears, and these organs, which to this time had been excellent, were at once found deaf, both by the patient and those about him.

Observation 3: A Paris merchant, age 40 years, small, very robust, large head and large chest, unused powerful muscles, was always seated in his office, scarcely ever going out except by carriage. Last December, without known cause and in perfect health, was taken with the following symptoms: He was stretched in an armchair warming his feet and preparing to roll a cigarette when his wife saw him lean forward and fall on the left arm of the armchair without saying a word or breathing a sigh. Lifted up in a moment, his face was seen to be pale and covered with sweat. If his head was released, it fell to the left and resumed its flexed position. Soon nausea followed and then vomiting. He was thought to have had an attack of apoplexy, and the sick man was carried to his bed in a state of complete relaxation. Consciousness returned, but malaise was marked. Everything in the room seemed to rotate, and the patient said he was seasick. He crouched in his bed as if he feared to be turned, and the vomiting continued. Needless to say, free bleeding was done, and resin purgatives given—severe treatment. Little by little the patient quieted and erect posture was possible, though with a little uncertainty in the gait.

Meanwhile the ears had become the seat of violent and continuous noises. Hearing was soon diminished, and the sense which hitherto had been good was lost for conversation in a low voice. M. X. was unable to go to the theatre as before, and his public and private life underwent considerable change.

Observation 4: Quite recently Dr. Laboulbene, a member of the faculty, called Prof. Trousseau and myself in consultation to see a patient from the South. This man, still young, short, dark and very nervous, had suffered from several attacks of vertigo, with nausea and vomiting. The doctors who witnessed the attacks had considered them due to a cerebral congestion, and had combated them by bleeding, leeches and purgatives, but the repetition of the same symptoms had cast doubt on the diagnosis, and as the patient found that his hearing, hitherto perfect, had diminished, that the ears were full

of noise, that the gait was staggering, he came to Paris and we were to establish the integrity of all parts of the auditory apparatus.

The patient, otherwise very well, suddenly experienced something like an arrest of cerebral function. He was walking along the boulevard when he felt faint, everything revolved about him, nausea set in, the face became pale, perspiration stood out on his forehead and he had to lean against a wall to keep himself from falling. These functional troubles only lasted a few minutes.

We had to look up with care all the circumstances which could throw light on the diagnosis of this case, but it was impossible to arrive at anything satisfactory. And as the derangement of hearing was the only appreciable sequela of this cerebral condition, we had only to conclude that the changes which were revealed by these symptoms lay in the internal auditory apparatus.

Ménière then concludes that: *1.* An auditory apparatus, hitherto apparently sound, may suddenly become the seat of functional troubles consisting of sounds of variable nature, continuous or intermittent and these noises are soon accompanied by a more or less extensive loss of hearing. *2.* These functional troubles, having their seat in the internal ear, can cause symptoms considered cerebral, such as vertigo, dizziness, uncertain gait, reeling; they are, moreover, accompanied by nausea, vomiting and a state of syncope. *3.* These symptoms, intermittent in type, are directly followed by deafness, slight or great, and often the hearing is suddenly and completely lost. *4.* Everything points to the belief that the material lesion which causes these functional troubles lies in the semicircular canals.

During this same year he mentions the following case, on which his diagnosis was made and on which, I think, most medical men have since that time become very much confused: A young girl, who, traveling on a winter night on a diligence, experienced as a result of a considerable chill, a complete and sudden deafness. Received on the service of M. Chamel, she showed as the chief symptom, continual vertigo, the least effort to move producing vomiting. Death occurred on the fifth day. Necropsy showed that the cerebrum, cerebellum and spinal cord were absolutely free from any change. The semicircular canals were filled with a red plastic substance, a sort of sanguineous exudate, of which only a few traces were found in the vestibule, and which did not exist in the cochlea. The most careful investigation enabled me to establish, with all the accuracy desired, that the semicircular canals were the only parts of the labyrinth that showed an abnormal condition, and that consisted, as I said, of the presence of a reddish plastic lymph.

Now, it would seem that this case today would probably be diagnosed correctly as a form of meningitis, an acute situation, while the other cases cited above might be termed, "Syndrome-Complex, Ménière, or the real type, a chronic disease. Thus I suggest that the expression, Ménière's Disease, be changed to Syndrome-Complex, Ménière, or Symptom-Complex, Ménière.

The nature of the change in the labyrinth has been necessarily a matter of conjecture. Politzer mentions that cases have died after presenting all the clinical features of the disease in which the postmortem findings have failed to provide any evidence of hemorrhage into the canals, and, on the other hand, an hemorrhagic exudate within the vestibule and canals has more than once been observed postmortem in cases where no history of typical symptoms during life could be obtained. Furthermore, this lone case, upon which our conception of the disease still very largely rests, does not supply a logically complete picture, for one of the conspicuous clinical features of the attack was the sudden and profound deafness, yet his postmortem examination revealed no abnormality within the cochlea.

Anatomically, the labyrinth is a closed cavity. Within this bony structure is the membranous labyrinth, a membrane so extremely sensitive to any form of direct mechanical injury that it is difficult to believe that a considerable exudate of any sort could fill even a portion of this cavity without causing practical destruction, or at least permanent ablation of function of the membranous structures contained therein. It is one of the inconsistencies of our conception of the disease that while the semicircular canals are looked upon as the characteristic site of the lesion, permanent deafness is said in severe cases to be one of the most constant and characteristic sequelae.

Stimulation or irritation up to destruction of the labyrinth or of any portion of the vestibular tracts induces vertigo or confusion, with the associated loss of equilibrium. Therefore, in diseases of all these regions vertigo is to be expected by at least implication of the vestibular nerve. Thus the character of the accompanying phenomena, especially the nystagmus, aids in the localization. This does not mean that irritation of the ear itself is the sole and only way of producing vertigo. We all realize that various visual disturbances, cardio-vascular affections, gastric or alimentary disorders, may exhibit dizziness as a symptom. However, in the latter instances it is the direct action on the vestibular apparatus that is responsible for the induction of the vertigo. The stomach itself, or any of the other organs, excepting the labyrinths, can no more produce vertigo than they can produce sensations of flashes of light or obsessions of smell.

The symptoms of irritation vary according to the intensity of the affection. These usually appear in paroxysms at irregular intervals. In the intervals, which may be weeks or months, the patients are either free from all disturbances of equilibrium, or there are sensations of dizziness and unsteadiness of gait, which may be more or less marked, and which do not disappear entirely. The attacks return either without any apparent cause or from bodily exertion, rapid turning of the head, bending or rising suddenly after a hearty meal, as well as after nervous excitement. A fresh attack is often preceded by increased tinnitus, a feeling of pressure and heaviness in the head, uneasiness, general depression and a sensation of rotation. The attack itself is, according to its intensity, characterized by severe dizziness, tinnitus and nausea, often accompanied by vomiting. When the patient endeavors to walk or stand, there is a tendency to fall forward, generally to the affected side. Even when quiet in bed with the eyes closed, the individual may have the sensation as if the entire room were revolving. Every attempt to change the position of the body or to rise increases the intensity of the attack.

Of the violent symptoms met at the beginning of the attack, the transitory unconsciousness and vomiting are the first to disappear, while the dizziness and disturbance of equilibrium as a rule diminish only in the course of several days to such an extent that the patient is able to walk a short distance, either with support or the aid of a cane. It is usually noticed that, when the patient walks, there is a tendency to fall forward, reaching for some indefinite object just out of reach. The disturbances of equilibrium experienced in the dark, and while walking with closed eyes, are especially noticeable, inasmuch as even in those cases in which the affection is of a mild degree, the unsteadiness of gait may be as pronounced as that met with in the most severe forms of ataxia. In addition to the above, it is found that in the first few weeks of the severer forms of deafness, psychical depression, slow cerebration and weakness of memory develop.

On close inspection of the patient it is noticed that the individual is usually of middle age, that the face is pale and bathed in cold sweat, while the subjective symptoms complained of are usually a bilateral, more rarely a unilateral, deafness, accompanied by severe tinnitus. The pulse is slow, the temperature may be subnormal and the nystagmus is a combination of horizontal and rotary. The vestibular nystagmus usually increases when the eyes are directed in the direction of the quick movement, and diminishes or ceases all together on causing the patient to focus in the opposite direction. The deaf-

ness may be of long standing or come on suddenly with the first attack. The loss of hearing usually remains stationary; it is only in exceptional cases that a slight improvement is observed. This may be permanent or again become lost in spite of active treatment.

Believing as we do that a definite percentage of these cases have an endocrine diathesis, it is my desire to cite the following cases, with an appropriate discussion regarding their outcome and suggestions as to therapeusis.

Case 1: Married man, age 56 years, successful merchant; chief complaints: tinnitus, vertigo, nausea and vomiting, with slight deafness. During the past 10 days he had five attacks, more or less severe in character; describing his sensations, he stated that as soon as he was able to sense his surroundings, objects in the room seemed to rotate to the right; that the window case, instead of moving directly to the right, moved with a wavy, dipping motion. That it seemed difficult to focus the eyes to near objects, as well as experiencing trouble in focusing on distant points. He had been extremely tired, sleepy and stupid, even after a full night's sleep. There had been no hiccup, but considerable gas in the bowels. The intestines had been sluggish, with some headache in the occipital region. There was a tendency to yawn throughout the entire time consumed in questioning and examining. He had a similar series of attacks seven years ago. The patient's family history showed heart failure, arthritis, one remote case of malignancy, several of gall stones, and apparently a very definite longevity. The patient recorded the minor ailments of childhood, diphtheria at 17, and a ruptured appendix five years ago. At times had experienced pain in the region of the heart. Takes an occasional liver pill on suspicion. Residual history not remarkable.

The physical examination showed good development and nourishment, hair sparse, heart and lungs negative, residual examination was substantially negative regarding the present condition. The urine was entirely normal except for the residual nitrogen fraction, which was high, a general indication of disturbed metabolism. Blood morphology showed a slight eosinophilia and a slightly high value for the endothelial leukocytes. The blood chemistry was normal. Serologicals were negative. Phthalein was normal. The sugar tolerance was depressed. The patient was 19 per cent overweight and with a fairly normal lung capacity. Basal rate was recorded as 18 per cent, with a slightly slow pulse. The audiogram showed a marked loss of hearing in the right ear, most noticeable in the lower frequencies. The Barany showed a slightly depressed reaction time, was otherwise

quite normal, as was the caloric test. The neurological examination showed that the reflexes were normal. Co-ordination was remarkably good for the lower extremities in the heel to knee, and heel along tibia. The upper extremities extended without tremor, there was no past-pointing of finger to finger or finger to nose. Extended hands were rotated evenly and at the same level. The cranial nerves were intact as tested, there were no parasthesias or anesthesias. The stations were correct with eyes closed and opened. X-ray showed a large sella with a doubtful thinning of the posterior wall, not a real evidence of pathology, with an unerupted third molar and a retained root. The endermal tests were negative. Eye examination showed somewhat enlarged blind spots and a slight field contraction. A Graham test by the oral route showed a pathogical gall bladder.

Diagnosis: Thyroid failure, possible liver complications.

Comment: This case showed physically a tangible gall bladder pathology, and as the X-ray made this seem likely, we have a sound explanation for the lowered sugar tolerance through the associated disturbed liver function. As a result of hypofunction of the thyroid, the assimilation limits for galactose are either normal or else raised above that which is essential to a normal metabolism. As a result of this lowered metabolism and temperature there arise the subjective symptoms of chilliness, cold extremities, and a desire for warmer or heavier clothing. In reality this instability may be due to the fact that at times the functioning of the thyroid gland is excessive, again it is deficient in the same individual. The eosinophilia may be due to the intestinal disturbance. In addition, the lymphoid blood, depressed blood pressure and pulse rate, together with the high residual nitrogen, determine the diagnosis of thyroid disease. The treatment instituted was thyroid extract, 2 gr. daily and increased in two weeks to 3 gr. daily with the meals. The subsequent notes showed that the basal rate returned to normal, the blood pressure remaining between 124/84 to 130/80; pulse, temperature and weight improving, the only thing complained of was the fatigability. He was troubled with insomnia at times, but this was probably due to the pressure of his business, as it was very demanding. It is now over two years that he has been under intermittent observation, taking 4 gr. of thyroid daily, and without any return of the vertigo, is free from all tinnitus and his hearing considerably improved.

Case 2: Married man, age 52 years, and for years had worked in a quarry. His chief complaints were violent vertigo, falling to the ground, deafness, tinnitus (throbbing type), nausea and vomiting. During the last year the periods of vertigo, their onset apparently

independent of eating, would incapacitate him, as on walking, either in the house or outdoors, especially if in the dark, he would fall prone, or if able to protect himself by a chair he would then gradually clear mentally, but on any quick change of position the vertigo and nausea would again make themselves felt. The description of the vertigo was generally of a mixed type and he was unable to determine the direction of objects, but was sure that either closing or opening the eyes made no difference on the confusion. Outside of general nervous tendencies the family history was negative, while the personal history showed malaria in his younger days, as well as mumps and measles. He smoked a great deal and formerly used alcohol excessively. Had two living children, who were well.

The physical examination showed a symmetrically developed man with a general loss of weight, color pale and the muscles seemed to be very rigid. The eyes reacted to light sluggishly but the pupils were equal. Ears showed advanced adhesive processes in both middle ears. The static labyrinth showed a normal reaction time and the caloric test was normal. The urine showed a slightly depressed amount for 24 hours, with albumin, but no casts. The blood showed nothing remarkable either morphologically or chemically. The serologicals were negative, as was the result of a spinal puncture. The phthalein was normal. X-ray showed fibrosis of the lungs and pyorrhea about all the remaining teeth. The gastro-intestinal examination showed no pathology. The neurological examination was entirely negative except the loss of the knee jerk on the left. The eye grounds were negative, as were the fields of vision. The basal rate was within the normal range (slightly raised), with a subnormal temperature and pulse rate. The blood pressure showed 142/84.

Diagnosis: A disturbance of the central nervous system (toxic) without any demonstrable endocrine involvement.

Comment: Treatment in this case consisted of extraction of all the teeth in both jaws, a change of occupation, together with supportive, outdoor treatment. This man improved very much indeed, so that six months following the first observation, although the deafness and tinnitus had not improved, the vertigo and nausea were absent and the dark did not trouble his locomotion. He had, however, an early interstitial nephritis, which is slowly progressive. He has been free from the peculiar somnolent attacks following the severer attacks of the vertigo that he experienced two years ago. The greatest diversity of symptoms, seemingly unrelated and not referable to any common cause, are encountered in all hypofunctional disturbances, even when a diagnosis of a specific gland involvement

cannot be made, thus a nonendocrine diagnosis is in order. This case was probably of toxic origin.

Case 3: A married man, age 68 years, retired from business, complained of marked sinking, dizzy attacks, headache (parietal and temporal), tinnitus, nausea and vomiting. During the past eight years has had several attacks, but during the last two winters the attacks have been very bothersome, due to their frequency. Before the attack, tinnitus increased in both ears until he seemed to hear nothing but the roaring, then he became confused and laid down. Several times on not lying down at once he has fallen. Objects seem to rotate in several directions and it makes no difference to the sensation whether the eyes are open or closed. The vertigo is apparently relieved after vomiting a number of times, but the depression and lassitude left as residua are very distressing. He also suffers intensely from seasickness. There were several cases of mental disturbance in the family history, which is otherwise negative. There was also a personal history of long standing constipation and a watery discharge for several years. In addition to the minor ailments of childhood the patient had scarlet fever, two operations for hemorrhoids, repeated probing of the right lacrimal duct many years ago. Had also pain in the high cervical region when doing hard physical labor.

The physical findings showed edema under both eyes, with numerous red nevi on the body. The occipital region showed a slight depression but was negative to the X-ray. The routine examination of the urine showed fair elimination, with poor balance. One sample showed albumin but no casts were found. There was a normal nitrogen partition. Phthalein low normal. The galactose tolerance was normal, with a normal shaped urea curve and a normal salol reaction. The blood showed a 3 per cent eosinophilia in a slightly lymphoid blood, while chemically there was an increase in non-protein nitrogen and uric acid. The serologicals were negative. The basal rate initially was —13 per cent—a value above the truth—with definitely slow pulse and low blood pressure and slightly low temperature. The alveolar carbon dioxid was normal, the patient was 9 per cent underweight. The Barany, while showing normal reaction time, elicited the same vertigo as the patient experienced before entering the hospital for study. The X-ray studies, endermal tests and eye examinations were all negative.

Diagnosis: Hypothyroidism (mild).

Comment: He had a high blood uric acid, but this loses much of its significance when it is associated with an increased nonprotein

nitrogen, which he showed in his blood repeatedly. The lowered basal metabolism would equally be referable to the patient's obviously low nutritional level, but with him this was not the case as repeated tests at two-week intervals gave levels of the metabolic rate distinctly lower than the first one reported. His 3 per cent eosinophilia might arise from several conditions, but with him, we think, it was due to faulty intestinal action. The action of the thyroid in affecting the general metabolism of the body explains the diversity of symptoms arising from its lowered activity. Constitutional tendencies may be made manifest in the general systemic disturbance resulting from glandular failure. Moreover, it has been demonstrated that the single gland may manifest over-activities and under-activities at different stadia of its functional derangement. Dysfunctional types are also recognized, in which the complex function of the gland may integrate diverse activities of its several functional components. Lastly, hypofunction is sometimes described as general debility and is intimately related to the infections, both acute and chronic, to the lowered vitality following prolonged nervous and emotional strain, and, as stated above, to constitutionally lowered vitality.

As to treatment for this individual, he steadily improved under 2 gr. of thyroid daily with his meals. However, he found that he could not omit the drug from his diet without slipping into the former distressing symptoms.

Case 4: A widow of means, age 60 years, had quite constantly for the past three years or more attacks of vertigo, associated with a very unsteady gait. The use of a cane had been of no great help, she exhibiting a tendency to go to the right. She has fallen in the house, but never outdoors. She had marked loss of hearing during the past five years. During several of the dizzy attacks she had tired herself out by vomiting. For the past few years had frequent attacks of herpes and for the past year had been very constipated. She also complained of great fatigability with the least mental or physical effort.

The physical examination noted a dry skin with several small, brown spots scattered over the body. Her reflexes are active, but the gait is unsteady and sense of accommodation and locomotion were poor. The cranial nerves seem, however, to be normal for a woman of her age. The urine showed fair balance, with a high residual nitrogen. The blood is lymphoid in character, with a slight depression of erythrocytes. There is a loss of vital capacity, while the basal rate is —31 per cent, with a lowered pulse rate and a lowered blood pressure and temperature. The serologicals are negative, the phthalein totaled 53 per cent.

Diagnosis: Hypothyroidism.

Comment: While all of the points presenting are not strictly in accord with the classical picture, the fatigability, constipation, hypothermia, high residual nitrogen, lymphoid blood and lowered basal rate leave but little question of a correct diagnosis. It is rare, however, that all the symptoms and laboratory findings are present in any one individual. Fatigue, disinclination for mental and physical work, particularly in the morning, are characteristic. The subject of hypofunction is never fit, he or she finds work a burden, and whether the employment is physical or mental, a push is required before the task is consumated. General nervousness and irritability are common symptoms, while constipation is very usual, due to deficient muscular movements of the bowels. The case was treated with 3 gr. of thyroid daily for several weeks. The vertigo ceased, but as she remained unsteady in her gait the thyroid was increased to 5 gr. daily. This helped her basal rate, bringing it up to —23 per cent, but otherwise she was not feeling as well as she thought she ought. The thyroid was omitted and thyroxin 1/320 gr., twice daily, was used. This seemed to make her better and stronger, allowing her to walk about her place without any aid. The appetite was improved. The thyroxin was increased to three tablets daily and now she is feeling very well, head clear, no vertigo, and can do as she pleases.

In commenting on the outstanding symptoms of the above series it is suggested that the giddiness is caused by the stimulation of the nerve fibres, rather than a diminution of their function. The tinnitus seems to be heard in both ears, but is almost always louder in one than in the other. It has been observed that the perception of musical notes is faulty in many of these cases. In the severer form nausea and vomiting follow the vertigo, and the pallor and physical depression are marked. Movements of the head seem to increase the confusion. After the attack the patient suffers for a few days from slight persistent vertigo. The tinnitus may or may not be increased by the paroxysms of vertigo. The attacks are sometimes accompanied by ocular disturbances, such as double vision, jerky movements of objects, and distinct erroneous projection in the direction of the movement, so that if the patient attempt to touch an object the hand goes too far in the direction of that object.

The attacks frequently come on suddenly, so much so that habitual sufferers are afraid to go upon the street alone; they are paroxysmal, and may occur daily or at much longer intervals, vague sensations being observed in the intervening period. These paroxysms may come on spontaneously or be induced by some sudden movement or

passing excitement. In the worst cases unusual intellectual effort may determine the attack. They may continue until the impairment reaches complete deafness, when there is usually a cessation of all confusion.

In partial or circumscribed disturbance of the vestibular end-organs in the labyrinth the vertigo is associated with nystagmus. The nystagmus is spontaneous and shows a long, slow movement, due to the vestibule, and a quick return movement, due to the tegmental nuclei, the direction of the quick movement naming the nystagmus. If the nystagmus movement is rotary and horizontal, it must be determined whether it is peripheral or central. A peripheral nystagmus to the right should show on caloric, pressure and rotation tests that the right vestibule is functionally active. Should such tests show an inactive right vestibule, then the nystagmus must be of central origin; if the right vestibule is active, then continued observations of the nystagmus will alone determine. Should the nystagmus continue uninterruptedly for 24 hours or more, it is of functional origin. If it lasts for shorter intervals and is interrupted by quiet intervals, it may be either peripheral or central. When there is also nystagmus of the well side, which lasts about two weeks, gradually decreasing, then a peripheral disturbance seems certain, as intracranial nystagmus is not so apt to diminish.

In a careful study of over 500 consecutive cases, that on laboratory examination were shown to be frankly endocrine in origin, and considering only the three dominant symptoms of the syndrome, namely, deafness, tinnitus and vertigo, I found these symptoms as not only present but, rather, the chief symptoms of complaint, in 289 pituitary, 148 thyroid cases and 119 gonad cases, as well as in 12 cases of adrenal involvement. This might be shown in the following table:

TABLE I.

Observations in an Endocrine Group.

	No.	Deafness	Tinnitus	Vertigo
Pituitary	289	22%	36%	24%
Thyroid	148	18%	34%	15%
Gonad	119	13%	34%	22%
Adrenal	12	25%	16%	33%

In another group study of a similar consecutive series of 500 case reports, shown to have a definite nonendocrine etiology, similar data are given in detail in the following table. The Miscellaneous column is composed of cases showing types of diseases involving the blood, bones, gastro-intestinal tract, respiratory system, eye, ear, etc.

TABLE II.

Observations in Nonendocrine Group.

Diagnostic Group	No.	Deafness	Tinnitus	Vertigo
Cardiovascular	61	24%	19%	40%
Neuroses and Psychoses	60	16%	11%	45%
Syphilis	43	18%	18%	32%
Focal Infections	56	28%	18%	23%
Lesions of the Central Nervous System	82	22%	13%	37%
Miscellaneous	198	39%	20%	22%

SUMMARY.

I do not wish to convey the idea that all cases of this interesting syndrome are endocrine in origin or that I have a panacea as to treatment, but I do feel that by no means all of these cases are surgical and that when by close laboratory and clinical study an endocrine diagnosis is established it will be found to be hypofunctional in character and never hyperfunctional. Again, I would like to point out that it is apparently the nutritional level of the individual that determines an increase or decrease of the presenting symptoms in this syndrome, especially in those cases of endocrine origin.

CONCLUSIONS.

1. As with every disorder depending upon an organic lesion, the severity of the symptoms varies with the extent and character of the lesion.

2. That attacks of labyrinthine vertigo are caused either by increased or decreased intralabyrinthine pressure.

3. That the syndrome-complex may occur in patients whose ears are already affected with deafness not of nerve origin at the beginning, and that the onset of deafness, on the other hand, may originate with the first labyrinthine attack.

4. That the autopsied case described by Ménière was an acute infection, probably meningeal in origin, while the cases of the syndrome-complex are always chronic in character and are directly of ear origin.

416 Marlboro Street.

DIAGNOSTIC AND THERAPEUTIC VALUE OF IODIZED OIL IN CHRONIC PURULENT OTITIS MEDIA AND CHRONIC MASTOIDITIS. PRELIMINARY REPORT.

DR. M. JOSEPH MANDELBAUM, New York.

The therapeutic use of iodine for diseases of the middle ear is not new. Its use, however, for diagnostic study of affections of the middle and inner ear, as far as I can find, has not been published, with the exception of its recent mention by Pitkin[1] and Spielberg[2].

Pitkin states he attempted to make a Roentgenographic study of the contour of the Eustachian tube by injecting iodized oil into the pharyngeal orifice of this canal by means of the eustachioscope.

He informed me (personal communication) that he repeated this procedure in the same manner in another case. Both these patients had intact eardrums and gave such violent reactions, intense dizziness and pains, that he refrained from repeating the procedure.

However, Spielberg previously obtained satisfactory radiograms of the Eustachian tube by injecting 10 to 15 drops of 40 per cent iodized oil into its pharyngeal end, drop by drop, through a No. 4 silk catheter inserted through a pliable silver Eustachian catheter. He reports no ill effects resulting from the injected oil, and mentions one case in which the presence of the iodized oil was demonstrated by the Roentgenograms taken one week after the initial injection.

The violent labyrinthine reaction, noted by Pitkin, was absent in Spielberg's work and probably was due to slowly instilling the oil, drop by drop.

Therapeutically, iodine has been administered hypodermically, orally and topically for its apparently selective local effect in diseases of the ear, nose and throat.

Lederman[3], in 1917, wrote enthusiastically of the results obtained by the use of a new iodine powder (Sulzberger) for treatment of chronic suppurative otitis cases.

The preparation, the exact formula of which has not been disclosed by its maker, is stated to readily liberate the iodine content. In a

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recent personal communication, Lederman states: "Since my original paper appeared, I have employed this treatment in cases of prolonged chronicity (one of 35 years' duration) and in cases in which radical surgical treatment was advised by other aurists, with equally as good results."

The iodine powder is introduced well into the external canal by insufflation and by means of compressed air, some of this powder is blown more deeply into the tympanic cavity through the perforated drum membrane.

Ira Frank[4] has recently verified Lederman's reports, stating he has been using iodine powder for over four years with unusual success in cases of chronic suppurative otitis media. He writes: "I have seen granulations disappear, suppurations cease and fair-sized perforations close. We are doing fewer radical mastoids each year. Recently I have tried it on subacute suppurative otitis media with the same good results."

Stokes[5] advocated the insufflation of equal parts of bismuth and iodoform for the treatment of chronic suppurative ears.

McKenzie[6] recently reported satisfactory results with iodine given in minute doses by mouth for head colds.

Sternberg and Goldberger[7] a few months later report similarly satisfactory results with small doses of iodine in catarrh of the middle ear. They injected subcutaneously 1 c.c. of a 5 per cent solution of sodium iodide, containing traces of free iodine, in patient with acute catarrh of the middle ear. They noted improvement within 24 hours, and recovery within six days in five out of 10 patients.

Sturm[8] also reports excellent effects from the use of "nascent" iodine in otorrhea. He states that "in both acute and chronic cases of middle ear suppuration better results than were obtained with any method of equal simplicity at present available; even in acute mastoiditis, where operation, though clinically urgent, was refused by patient, it has cured apparently desperate cases". His method consists in the internal administration of potassium iodide with local applications of hydrogen peroxid drops. An adult patient takes 10 grains of potassium iodide, well diluted, every four hours; every two hours, or even more frequently, the external meatus is filled with hydrogen peroxid, which is retained for 10 minutes. The resulting reaction between the absorbed salt and the applied drops liberates nascent iodine within the tissues of the infected middle ear cleft. "With variations of the dosage of the active salt, according to the age and idiosyncrasy, the treatment is useful in every case of suppurative otitis media."

The publication in 1922 of Sicard and Forestier's work[9] on the intraspinal injection of a new preparation of iodine in vegetable oil for diagnostic purposes has been followed by the injection of iodized oil into almost every bodily organ and recess. The comparative impunity with which this oil has been injected and the remarkable tolerance of the most delicate tissues to the preparation led various workers to adopt it for the diagnostic study and functional investigation of the various bodily systems.

Following the original article on myelography, iodized oil has been used in the bronchial tree, the pleural cavity, the paranasal sinuses, the Eustachian tube, the female genital system, fistulous tracts of all kinds, and for many divers purposes.

During the past two years while having under observation a number of cases of chronic suppurative otitis media of recurrent type in which there occurred periods of prolonged subjective mastoid sensitiveness and in which a comparative Roentgen study of both mastoids failed to elicit satisfactory information, I decided to investigate the value of iodized oil as a shadographic medium in a Roentgenodiagnostic study of diseases of the middle ear. Fortunately I was unaware of Pitkin's experience from injecting the oil into the pharyngeal orifice of the Eustachian tube, as knowledge of the untoward symptoms following this method might have deterred me from making my investigations.

The following cases are typical of a comparative study of the mastoids of each patient after the injection of the iodized oil.

Case 1: L. L., male, white, age 34 years, first presented himself for examination on Nov. 18, 1926. Twenty years ago suffered with a running left ear, which he believed due to a cold. A spontaneous rupture of the drum took place. Ear was irrigated frequently, following preliminary injection of peroxid of hydrogen by his physician. While treatments were very painful, he recovered in one week. The hearing in this ear, however, remained subnormal ever since this attack. Did not have a recurrence until 18 months ago, when while swimming got water in left ear, which without any pain was followed next day by discharge of a watery, yellowish-colored serum. He washed the ear with a bulb ear syringe twice daily with warm water for three days, when the discharge ceased. One week later and every week or two thereafter, following swimming, the discharge would recur without pain and the same treatment seemed to relieve it. In the fall of the year after the swimming season, the ear ceased discharging and he was well during the winter of 1925, excepting when he drove his automobile with a direct draft against left ear, the dis-

charge would recur, being of the same thin, yellow, serous character and unaccompanied by pain. For these attacks during the winter he would also syringe the ear with plain warm water. They occurred about once monthly until the summer of 1926, when he had the same experiences as the previous summer. In October, 1926, caught a cold, with recurrence of the same discharge. Then consulted an otologist, who prescribed acriviolet solution, to be instilled into the left ear twice daily. The patient failed to carry out the orders, using plain warm water instead, which again was followed by recovery until Nov. 11, 1926, when without any ascertainable reason, apparently not having caught cold nor exposed himself to drafts, the ear suddenly began to discharge a profuse blood-stained, sticky mucoid discharge. This was unaccompanied by chill, fever, pain or dizziness, but had a "dull" sensation over mastoid.

He again syringed his ear twice daily for a week, which was the longest period the discharge ever had continued. Today, Nov. 18, 1926, upon his first visit to me the ear was still discharging but he thought somewhat less. The examination of the left ear revealed the external auditory canal filled with a thick mucoid discharge, the pledget of cotton which he had placed into the ear several hours previous being saturated with the secretion. After removing the secretion in the external canal by suction the drum appeared to be totally destroyed, the tympanic cavity highly injected and a pulsating light reflex noted upon some deeply situated secretion. Only a small triangular remnant of drum membrane remained attached to the long process of the malleus.

Some small granulations were noted in the upper posterior quadrant. The auricle was extended slightly outward and forward from its attachment to the head; a small area over the mastoid antrum was senstive to pressure, as was the tip. The usual tests for hearing showed the presence of a chronic left catarrhal deafness. There was no evidence of any labyrinthine disturbance, as shown by the caloric and turning reactions.

The right ear was normal and the nasopharynx showed no marked pathology.

A smear and culture of the discharge of the left ear was made by Dr. J. Pincus on Nov. 18, 1926, with the following report:

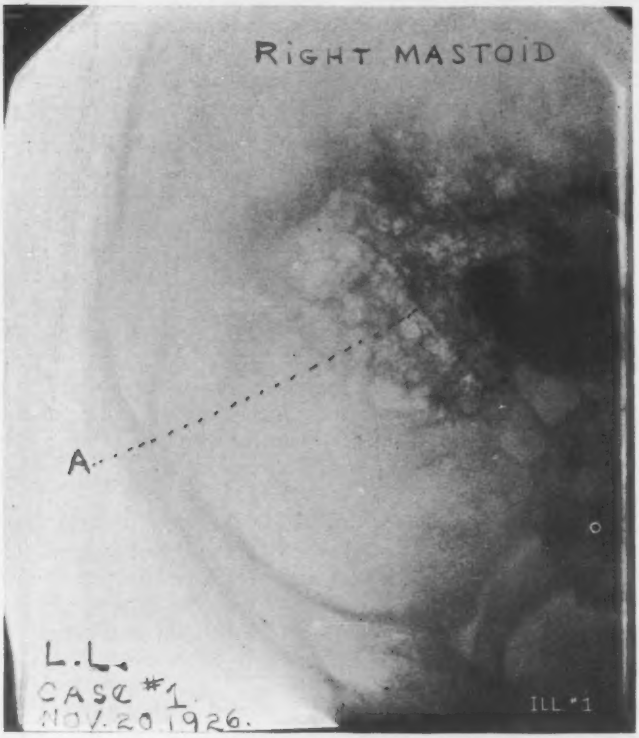
"Smear shows many pus cells and Gram-negative encapsulated diplobacilli and diplococci.

"Culture: Bacillus mucosus capsulatus."

Clinical Diagnosis: Acute exacerbation of a chronic recurrent left otitis media purulenta associated with a subacute mastoiditis.

A Roentgen study of both mastoids was made by Dr. S. Fineman on Nov. 20, 1926 (Figs. 1a and 1b), with the following report:

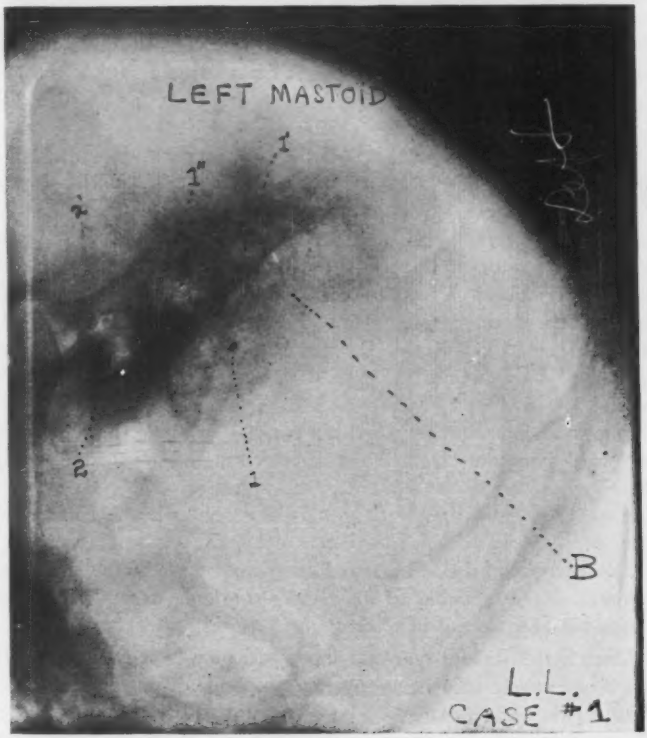
"The two mastoids are asymmetrical. The right mastoid is apparently developed and well pneumatized. The left mastoid is smaller and shows a considerably smaller amount of cell structure in its posterior portion and practically no cells in the region of the zygoma.



The position of the anterior wall of the two sinuses, while not exactly symmetrical, is normal.

"The right ear shows a large, well pneumatized mastoid in which the small cells surround the antrum and extend posteriorly to the sinus and anteriorly into the zygoma. The cells behind the sinus are considerably larger and show no periosteal thickening or cloudiness.

"The left mastoid shows a slight diffuse cloudiness of all visible cells. The sinus is much more distinct than on the right. In the lower portion and in the tip of the mastoid there are a number of irregularly-shaped air cells which very strongly suggest a loss of septal structure. In addition, in the upper and most posterior portion of the mastoid, there is a very large area of rarefaction, which may or may not be an area of broken down cells. This area seems to lie just



a bit too far out for one to be certain about its definite relationship to the mastoid. In addition, no cells are to be seen extending into the zygoma.

"Conclusion: The left mastoid is smaller than the right, due probably from sclerosis resulting from former infection, and gives evidence of definite disease. It is not possible to tell, of course, from the plates alone, whether the disease is acute or of old standing, but the

X-ray does show evidence of a past or present pathological process in the left mastoid."

Patient was treated daily for a few days by removing by suction the secretion from left external auditory canal and tympanic cavity. The discharge became so profuse that frequent irrigations with warm normal saline solution were necessary. Various antiseptic solutions were used without success, including the "ether" treatment. After two weeks, as the tender area over the mastoid antrum became slightly reddened and he developed an uncomfortable feeling over the entire left side of head, and a fever of 101.5° F., with the previous bacteriologic report of bacillus mucosus capsulatus, a radical mastoidectomy was considered. Having in mind, however, the possible diagnostic value of iodized oil in cases of this type, and the entire absence of the eardrum making its instillation a simple matter, I decided to utilize this medium.

Technique: Patient was instructed to instill 2 c.cm. of ether into affected ear morning and night, with affected ear uppermost; after 10 minutes, sufficient time for the ether to evaporate completely, 1 c.cm. of iodized oil was injected daily into the external auditory canal of the affected ear, holding the affected side uppermost. He was instructed to lie in this position 10 minutes longer, to allow the iodized oil to penetrate by gravity as deeply into the inner ear as possible.

One week later, during which time, not only did he not suffer any untoward symptoms from the treatment, but the purulent discharge ceased to appear his mastoid pain disappeared.

Another Roentgen study (Fig. 2) was made by Dr. S. Fineman on Dec. 20, 1926, with the following report:

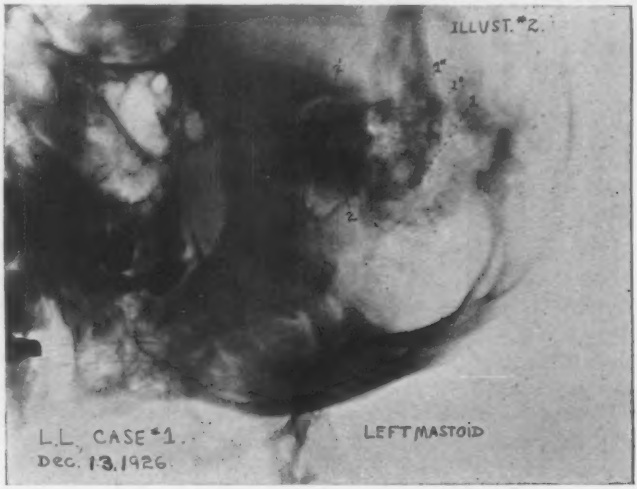
"No changes in the left mastoid other than those of the last examination. *A few globules of an opaque substance are seen scattered within the antrum and in some of the mastoid cells.*"

Nov. 1, 1927, about one year later, patient was re-examined. He had been entirely free from any ear symptoms during the past year, neither pain nor discharge. The external auditory canal of the left ear was free from any secretion, the inner wall of the tympanic cavity was covered with smooth, dry, glistening, healthy-appearing mucosa. The hearing had not improved. Patient was discharged as cured and has remained well since.

Case 2: First seen by me Sept. 14, 1927. R. W. W., female, white, age 38 years. Chief complaint: Pains about right parieto-occipital region. Had measles at 6 years, followed by right mastoiditis. Mastoidectomy was performed. Quite well till 12 years of age, when

there was a spontaneous opening of the mastoid wound, with a discharge of pus. The surgeon who operated upon her made a diagnosis of erysipelas, and sent her to the hospital, where she recovered completely in five days. Was well until 17 years of age, when a discharge of the right external auditory canal was noticed, which ceased after two days. Remained will until 25 years of age, after which she had an occasional discharge, first thick in quality, but in a few days would become thinner and then disappear. These attacks recurred about once yearly until four years ago.

In February, 1923, her general health became poor and she began to develop severe head pains at night about the right occipital region.



These continued until she was operated on in Philadelphia by another otologist, who drained the mastoid through the posterior wall of the external auditory canal. The original wound was not opened. He found a necrotic area and drained it. It was 2½ months before the wound healed.

The pains disappeared and the patient seemed well until eight months later, when, in a Western city, she was advised to have her tonsils removed as the pains in her head had returned. It took all winter to recover from the tonsil operation, owing to general debility.

In October, 1924, she developed a urological condition, which was diagnosed as a tuberculous kidney. During this period she had bloody

urine and painful urination. The symptoms continued for three weeks. Patient had X-ray and cystoscopic examinations and tuberculin treatment, and all symptoms disappeared. Weight increased and felt well until one month later, when pains again developed in head, covering entire right side of cranium. Roentgen examination of the skull was negative and was told the pains were "reflex". Was in hospital one week when recovered.

In February, 1925, returned to Philadelphia, having been told to continue the tuberculin treatment. But the physician she was referred to doubted the diagnosis and would not continue its use. She was in fairly good health until June, 1926, when had a complete breakdown, during which period the ear would discharge at times. Was again cystoscoped. Examination negative and no Tbc found. But was again given tuberculin treatment, following which she seemed to improve, although slight aural discharge occasionally recurred. Moved to New York and physician to whom she was referred to used the sun-ray lamp treatment for the discharge, which at first was seemingly controlled by this means, until Sept. 14, 1927, when the ear again began to discharge more profusely. She was then referred to me by her family physician.

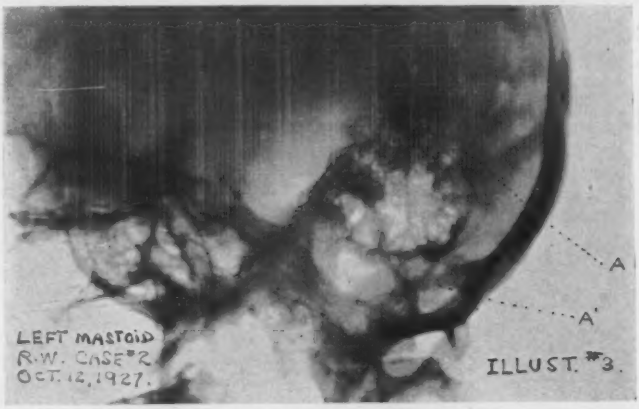
Examination of the patient's right ear revealed a well healed mastoidectomy wound. No tenderness over the mastoid antrum, tip or emissary vein, but at a point over the extreme angle of the postero-superior limitation of the mastoid cells, corresponding to about the union of the parietomastoid and occipitomastoid sutures, was a small tender area which was slightly sensitive to pressure, but patient stated that the pain seemed "deep in". There was some pain upon pressure over the right suboccipital region. There was a slight amount of odorous, thick, yellowish discharge seen on the floor of the external auditory canal. This passage was somewhat stenosed and extremely deviated, apparently a result of the previous operation through the posterior canal wall. Upon wiping away the discharge it was noted that the annulus was very small, the drum practically destroyed except for a narrow antero-inferior crescent-shaped remnant, the long process of the malleus seemed intact and at the postero-inferior quadrant could be seen the anterior border of the foramen rotunda.

Culture of the discharge was taken, as well as a smear. No Tbc found. Tuning fork tests revealed no response of the right ear to either air or bone conduction and the usual voice tests disclosed absolute deafness. There was no nystagmus, past-pointing or other signs of labyrinthine disturbance. The left ear responded normally. Wassermann test negative, as was the blood count and urinalysis.

Clinical diagnosis: Chronic otitis media purulenta; chronic mastoiditis following incomplete mastoidectomy.

The satisfactory results obtained previously in a number of similar cases from the use of iodized oil led me to utilize it in this case also. Without any other treatment whatsoever, the iodized oil was used at once exactly as described in Case 1. Patient began to improve almost from the first injection. The occipital pains gradually diminished, the otorrhea subsided, and on Oct. 25, 1927, the discharge had entirely ceased for over a week. A Roentgen examination (Figs. 3a and 4b) was then made by Dr. S. Fineman on Oct. 31, 1927, with the following report:

"The left mastoid shows a very large number of clear cells of varying sizes. The majority of the cells are small.



"The right mastoid shows the cells in the outer half of the mastoid to have been removed, apparently by operative procedure. A few fairly large cells, extending from the upper border to the tip of the mastoid, are still present. The area surrounding the antrum looks cloudy and shows no cell formation. However, *a large number of lipiodol shadows are seen scattered throughout this dense area* around the antrum and in some of the cells of the tip of the mastoid. The lipiodol discloses the presence of air spaces around the antrum region and in the upper part of the mastoid, 'which ordinarily could not have been visualized without the aid of the lipiodol'.

"I do not know whether these lipiodol globules lie in the mastoid cells or merely in the spaces between fibrous strands which might form after operation."

Case 3: First seen by me on July 27, 1927, S. O., male, white, age 45 years. Chief complaint: Deafness in left ear and occasional discharge. Past history: Has suffered from heart trouble for a number of years and was told he had angina pectoris; becomes easily fatigued and although he rests well at night, on awakening feels tired, but gets stronger as the day goes on. Never fainted. Has had no operations nor accidents.

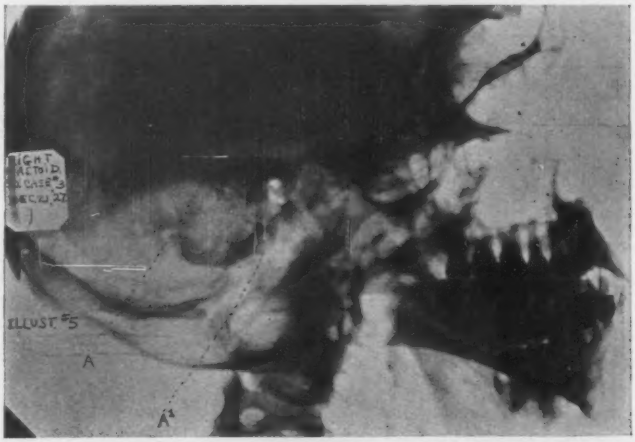
Present illness: Had a discharging left ear at 6 years of age. Following that, whenever he caught cold the ear would discharge. This

condition recurred until he was 26 years of age. Following which until the age of 45 years he was free from ear symptoms, excepting absolute deafness on that side. During that period he frequently went swimming; even this caused no trouble. Early in 1927, at irregular periods, he noted a slight discharge from the left ear about once a month, which would usually be noticed on awakening in the morning. It was thin, colorless, watery and without odor and would continue from 12 to 24 hours, unaccompanied with pain in the ear. He noticed an "itching deep in the ear" just before the ear would cease discharging.

During the past year had several attacks of sore throat and his family physician advised him to have his tonsils removed. I per-

formed a tonsillectomy on July 27, 1927, under local anesthesia and he made an uneventful recovery. This operation had no influence upon his otorrhea.

On Sept. 20, 1927, examination of the left ear disclosed the absence of the left eardrum and considerable amount of thin, watery secretion filling the tympanic cavity and external auditory canal. Upon removing the secretion by suction, the inner tympanic wall was exposed. It was reddened, covered with a few superficial granulations and the malleus was missing. He failed to respond to any of the hearing tests and gave no symptoms of labyrinthine disturbance. There was no tenderness over the mastoid.



Clinical diagnosis: Recurrent otitis media. Chronic sclerotic mastoiditis.

Patient was instructed to use the iodized oil in the same manner as prescribed in the previous cases.

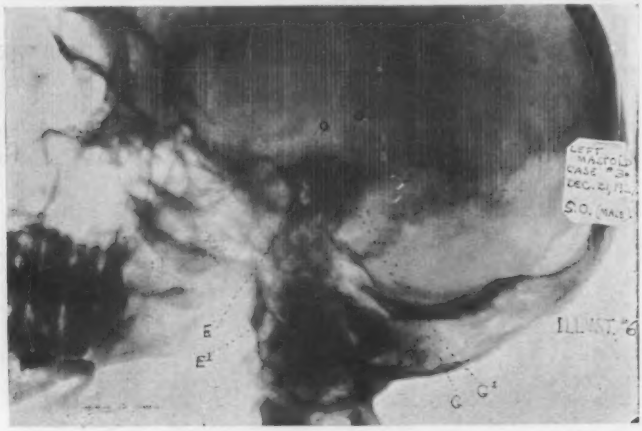
By Oct. 15, 1927, the discharge had entirely ceased and all subjective ear symptoms had disappeared. The use of the iodized oil was discontinued and until the present writing, Feb. 10, 1928, he has remained well.

A comparative Roentgen study of his mastoids was made on Dec. 22, 1927, by Dr. S. Fineman, which showed the following (Figs. 5 and 6):

"The right mastoid looks normal. It is of moderate size and presents a large number of well aerated small cells, most of which lie posteriorly to the antrum and in the region of the tip of the mastoid.

A few superficial cells are visible about the antrum. No zygomatic cells are to be seen. The lateral sinus and the emissary vein are clearly visible. The sinus seems to be wide and to be superimposed by the major portion of the mastoid cells. The middle ear looks clear.

"The left mastoid is of the infantile sclerosed type. The sinus and emissary vein are again visible, in an overlying area which corresponds roughly to the same area in the opposite ear. On this side, however, no cells whatever are visible over the sinus area. That portion of the mastoid which lies just anterior to the knee and sigmoid portions of the sinus, fails to show the same arrangement of aerated cells which is distinctly present on the right side. On this, the pathologic side, this area is on the whole cloudy and presents a



very few ill-defined spaces which might be interpreted as cloudy cells. These few spaces that I am referring to lie between the anterior wall of the sinus and the middle ear and within this area *are to be seen a number of lipiodol globules* which seem to bear a distinct relationship to the above-mentioned cloudy air cells. *A large number of lipiodol globules are also visible in the uppermost region of the mastoid,* extending all the way from the knee of the sinus to an area immediately above the middle ear. An elliptic, sharply defined shadow is visible in the region of the middle ear. I do not know exactly just what this represents. It is probable that this *represents the outline of the middle ear coated with lipiodol.*"

As stated at the beginning of this article a very careful search of the literature revealed that iodine has been administered hypoder-

mically, orally and topically for its apparent selective local effect in diseases of the ear, nose and throat and instances for its application for such usage have been quoted with references.

Since writing this article I noted another method of applying iodine for its therapeutic effect in a case of double suppurative otitis media which it would be well to quote here.

J. W. Torbett[10] reports as follows: "About 23 months ago my little niece, age 4 years, had an attack of scarlet fever, followed by profuse double suppurative otitis media which was treated by all methods by our specialist, Dr. E. P. Hutchings, who treated her for four months without results, and adenotonsillectomy and X-ray had been tried. I finally suggested the Ionic method of treatment with continuous current, but Dr. Hutchings said that would not do because the reaction and dizziness were too great on a child. I asked him where he placed the negative pole and he said on the arm. I suggested that he use a block tin, well padded, over the mastoid attached to the negative pole over the side being treated in the ear by the positive pole; this does not put any current in the internal ear, hence there was no dizziness or reaction whatever. The pad of the negative pole was saturated with a weak solution of about 5 per cent sodium iodide, from which the iodine ions are driven into the mastoid cells. A zinc wire connected with the positive pole was fixed on the inside of a rubber speculum placed in the ear so that the wire would not pass beyond the speculum. The ear to be treated was always turned up to be filled with ½ to 1 per cent zinc sulphate solution to which a little glycerine had been added. The current was given from a radio battery with 22½ volts controlled by a rheostat giving from 2 to 5 milliamperes—10-15 minutes. The little girl experienced no unpleasantness whatsoever, playing all the time she was taking the treatment, and had no reaction and took the treatment every other day. She has been perfectly well now for 1½ years, and a recent test showed her hearing normal. The thing new in the technique is the iodine solution on the negative pole and its application to the mastoid cells on the side being treated."

Conclusions: Since the use of iodized oil in cases in which it was proven that the oil globules penetrated to the uttermost residual cellular recesses of the mastoid cells, together with the surprising and satisfactory therapeutic influence of the iodine on the diseased deep structures, I have used it in more than 20 cases of "chronic" otitis, associated with varying degrees of subacute and chronic mastoiditis or acute exacerbations of chronic mastoiditis. with considerable success.

The purpose of this article is to suggest to other colleagues the use of this medium in cases of a similar type, to have Roentgen studies made of the injected mastoids before and after injection of iodized oil, and to report both their Roentgen findings and therapeutic results.

I wish it to be understood that in acute mastoiditis with distinct operative indications and in chronic cases with acute exacerbations calling for operative interence, iodized oil in the light of my present experience is *not* to be considered of any moment.

It has proven, however, that in several cases previously mastoidectomized, with recurrent attacks of mastoid pain and discharge, the presence of unremoved mastoid cells can be visualized, and in these few cases, at least, that the use of the iodized oil has apparently brought about a temporary cessation of pain and discharge and complete relief of symptoms.

The visualization Roentgenologically of the presence of unremoved mastoid cells by means of iodized oil, the recurrent or chronic infection of which are responsible for the continuation of middle ear disease, I believe to be of distinct diagnostic importance in cases of this type.

The unexpected therapeutic value of the iodine content of the oil in a number of these cases reveals additional advantageous possibilities.

I feel, therefore, that considerable benefit is to be derived from a further study of the use of iodized oil in cases of chronic otitis media purulenta and chronic mastoiditis from both the diagnostic and therapeutic standpoints.

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125 West 72nd Street.

TONSILLECTOMY—LOCAL vs. GENERAL ANESTHESIA IN RELATION TO LUNG ABSCESS.

Dr. William Lawrence Gatewood, New York.

A discussion of lung abscesses after tonsillectomy under local anesthesia is almost like a discussion of snakes in Ireland; there are very few reported. There are several reasons for this fact. Lung abscesses occur in relation to tonsillectomy by inhalation of blood or detritus or by dislodgment of emboli from the veins.

Under general anesthesia there is an opportunity for inhalation during the operation, which is necessarily conducted under deep narcosis in order to abolish reflex gagging, whereas under local anesthesia, properly administered, the reflexes of the larynx and epiglottis are maintained. Under these circumstances inhalation of foreign matter or blood may occur, just as inhalation of food occurs occasionally at meals when it goes down the wrong way. After operation under local anesthesia the swallowing reflexes are intact, the patient consciously protects the larynx while there is oozing, and the protective mechanism is reinforced by the pain of swallowing. Saliva and blood may be conducted from the mouth by posture and gauze seepage.

Myerson in making a bronchoscopic examination of 200 patients immediately following tonsillectomy, found blood and mucus in the trachea and bronchi in 155. This definitely proves that aspiration does occur under general anesthesia. In further support of this, Moore found that in pulmonary abscess following tonsillectomy the lower lobe of the lung was affected in 60 per cent, the right lung in 31 per cent, and the left lung in 19 per cent, which is the same relative proportion as that given by Chevalier Jackson in the aspiration of foreign bodies.

Subsequent to the operation under general anesthesia, the swallowing reflexes are abolished for a varying period. Unless the narcosis is deep, bleeding cannot be stopped; and if it is deep, the narcosis continues for a longer period than when it is light. During the after-narcosis the larynx is unprotected; if the patient is on his back, saliva or blood may flow into the bronchi; if on his belly, there may be vomiting and his condition is less well observed; the lateral

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postures for the head may incompletely protect the larynx from fluid in the esophagus.

So much for the mechanism by which the larynx permits the entrance of infected foreign matter into the lung. The entrance of uncoagulated blood is especially disastrous during this period of abolished reflexes, as it is not expelled promptly by coughing, and is permitted to coagulate, forming a web which neither the peristaltic action described by Bullowa and Gottlieb or the ciliary action can dislodge. Secretions accumulate behind the obstruction formed by the clot and the lung becomes eroded from retention and pressure.

This postoperative abolition of reflexes under general anesthesia is probably sufficient to account for the greater frequency of lung abscess. With proper handling after operation under local anesthesia the incidence of foreign body inhalation should be no greater than during meals, and with intact reflexes, blood should be promptly expelled before it is clotted, by coughing, peristalsis and ciliary action.

Some lung abscesses may result from the breaking down of infarctions. This may occur after local or after general anesthesia. A properly induced local anesthesia employing the infiltration method I have described, compresses the veins distal to the capsule and thus induces thrombosis in a clean and uninfected field. After general anesthesia the nausea and vomiting tend to dislodge thrombi and may induce bleeding from the arteries, or pulmonary embolism from veins, which are more apt to be infected if coming from infected field under general anesthesia.

It is obvious that whether we accept the inhalation or embolic origin of pulmonary abscess after tonsillectomy, the operation under local anesthesia mitigates the chances of lung abscess: on the one hand, by preserving the laryngeal reflexes during, and especially after, operation; and on the other, by aseptic compression of the veins in anticipation of the operation.

330 Park Avenue.

UNUSUAL CYSTS OF THE MAXILLA: CYST OF NASO-PALATINE DUCT; CYST OF FACIAL CLEFT AREA (FISSURAL CYST).*

DR. JOSEPH SCHEOFF, New York.

The two cases which I wish to present tonight are of unusual interest from several standpoints: *1.* Their infrequency; *2.* they border between the nose and throat and oral surgery fields; *3.* the usual dental classification of cysts of the jaws are inadequate. Both cases were first examined by nose and throat men, who referred them to the oral surgeon as dental cysts. As these cases could easily be confounded with dental cysts, which, however, have distinctive differences, it might not be amiss to first say a few words about dental cysts.

Cysts of the jaws are usually related to the dental system and are generally classified as: *1.* Root cysts; *2.* follicular or dentigerous; and *3.* multilocular or cystomas also called adamantinoma polycysticum). These cysts are found in the bone. Root cysts are by far the most frequent in occurrence. In the study of 416 cases, Rosenstein[1] found 394 root cysts, 13 follicular, and nine that could not be differentiated. Multilocular cysts, while considered rarer, are still found fairly frequently. I have seen four cases definitely proven by microscopic examination. The question as to whether adamantinoma should rightfully be called a cyst or a tumor with cystic tendencies would lead to unlimited discussion, far afield from our subject.

The root cysts are found in connection with infected or carious teeth, the process passing on to the pulp with the formation of the so-called granuloma at the apex of the tooth. In many cases these granulomas develop into cysts if epithelial cell rests are present in the peridental membrane of the tooth.

The follicular or dentigerous cyst is not associated with caries of teeth or infection of the peridental membrane, but rather with infection of the dental follicle of the developing tooth germs of the permanent teeth. They may arise from misplaced teeth anlage or supernumerary teeth. Hence follicular cysts may be found not only in the alveolar process of the jaws, but anywhere in the jaw bones. Cases

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have been reported in the palatine bone, maxillary sinus, ascending ramus of the jaw, etc. According to J. Lartschneider[2] the infection of the dental follicle is brought about through infection from neighboring persistent deciduous roots. His studies of 14 cases show that only roots of healthy deciduous teeth become absorbed. As the dentigerous cyst implies, there may be found within the cystic follicle either a whole or part of a tooth, depending upon the stage of development of the follicle when first infected.

The multilocular cyst or adamantinoma polycysticum arises as the result of continuous trauma or infection of persistent enamel organ epithelial debris in the jaws. These dormant epithelial rests becoming activated, proliferate and form tumors which later undergo cystic degeneration. The multilocular cyst type occurs more frequently in the lower jaw, while the solid type is found more often in the upper.

GENESIS OF DENTAL CYSTS.

From the genetic standpoint, all three types of cysts are associated with remnants of the enamel organ of teeth. The tooth germ starts to develop about the sixth week of embryonic life. It begins with proliferation of epidermal cells covering the jaw, which heap up to form a continuous dental ridge over the site of the future teeth. Soon some of the cells burrow down into the underlying connective tissue and fill the dental groove in the mesoderm. On the lingual side of the dental lamina thus formed, further proliferation takes place, giving rise to the 10 deciduous teeth buds. These teeth buds, resembling simple tubular glands, sink into the mesoderm. The proliferation of mesoderm cells at the time tends to invaginate these tubular glands. The invaginated bulb becomes the enamel organ, while the mesodermic cells form the dental papilla. The enamel organ thus consists of two coats or tunics—the outer and inner, between which are found stellate reticulum and stratum intermedium. From the inner tunic come the ameloblasts or enamel-forming cells. The dental papilla gives rise to the dental pulp and dentine of the tooth. Soon at the base of the enamel organ and dental papilla, mesodermic cells become modified into a connective tissue surrounding the tooth germ and form the fibrous sac—the dental follicle. The dental follicle grows from below around the papilla and, gradually progressing upwards, finally encloses the tooth germ and severs the cord from the enamel organ[3].

With the formation of the dental follicle and the cutting off of the epithelial cord, the stellate reticulum, the outer tunic and the epithelial cord having served their function, atrophy and tend to disappear. However, as Malassez[4] has shown, many of these cells do not

disappear, but persist throughout life and it is the activation of this "paradental epithelial debris" that give rise to the epithelial lining of the cysts. Malassez had demonstrated remnants of cells in the gums of infants, around roots of teeth, in the peridental membrane, etc.

Clinically, the root cyst is diagnosed by: *1.* The history; and *2.* the X-ray findings. Teeth with root cyst give a history of either trauma, with subsequent insidious death of pulps, or a history of caries, followed by some dental procedure, as root canal filling, etc. The X-ray finding will show an unsuccessful root canal filling or a retained root or granuloma that was not removed at time of extraction. Furthermore, such teeth when prseent will prove to be non-

Fig. 1. X-rays of two types of dental cysts in the upper incisor region. Left: History of infection of teeth with X-ray evidence of root canal fillings in the teeth involved in the cyst. Right: History of trauma to the lateral tooth four years ago. In both cases the teeth are non-vital and do not respond to the heat and the cold or to the electric pulp test.

vital, *i. e.*, they do not respond to the heat and cold or to the electric pulp test, etc. As a general rule, such teeth also show evidence of dental work or destruction of a portion of crown, etc.

The follicular cyst is usually found in the young and is slow in growth. It is frequently revealed by the asymmetry of the face that it produces as the result of tumor or swelling. The X-ray of the region confirms the diagnosis.

The multilocular cyst or adamantinoma cysticum is very slow in growth and is seen most often in young adults. The histories are somewhat similar, *e. g.*, following an extraction of a tooth, with curettement, the area does not heal, or if it does heal temporarily, it

recurs and is followed by curettement again, etc. Usually the X-ray discloses an area of large destruction of bone that has the appearance of multiple cysts.

CYST OF THE NASO-PALATINE DUCT.

Case 1: The case is that of a male physician, age 31 years. The history and examination was as follows: Patient was in good health. He complained of a strange feeling in the region of the upper lip and nostrils; slight fullness and tenderness was experienced when the upper lip was raised. Palpation over the areas and over the apices of upper incisor teeth elicited tenderness. Nasal examination was negative.

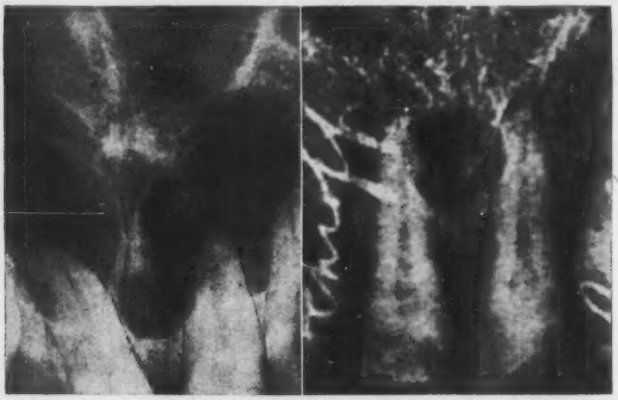

Fig. 2. X-ray of the cyst of naso-palatine duct. No history of dental infection or trauma. The teeth were vital.
Fig. 3. X-ray of the upper incisor region with an area of decalcification formerly considered to be an enlarged incisor foramen.

Teeth: All incisors present and in excellent condition. No evidence of dental work. They were normal in color, responded to heat and cold and to electric pulp tests. There was no history of trauma from the patient or from the parents, both of whom were living. X-ray revealed a large area of decalcification in median line, with apices of teeth (incisors and lateral) apparently within this area of decalcification. The area had the appearance of a cyst and lay between the floor of the nose and the alveolar process. It appeared as if there were two cysts, the larger being on the right side.

Before operation, the question of a diagnosis arose. Was it a root cyst or follicular cyst from a supernumerary tooth, which is common in this region, or was it something different?

The operative procedure through the mouth was considered better, although it was realized that the nerves of some of the teeth would, most likely, be destroyed. After elevating the mucous membrane on the right side, high above the apices of the teeth, there was seen directly underneath, the cyst membrane through the eroded buccal plate of the bone. The cyst membrane was then shelled out with difficulty. The membrane seemed to be held in the midline. Throughout the operation, the patient complained of pain in the naso-palatine region, especially when the membrane was tugged.

After the operation a probe could be passed through a hole in the median line of the maxilla from the right to the left side. The cyst

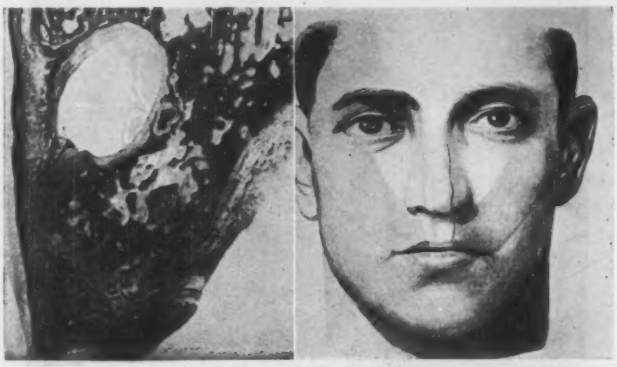

Fig. 4. Photomicrograph of the section through the jaw of Fig. 5, showing the so-called enlarged incisor foramen to be a cyst of the naso-palatine duct.

Fig. 5. Fissural lines of the face (Sicher and Tandler). Point X is the junction of the lateral and middle nasal processes and the upper jaw process. This is the site of the fissural cyst (facial cleft cyst).

contained a large amount of a thick, brownish-red inspissated material. (The descending palatine artery passes through the naso-palatine canal.) As we were under the impression that we were dealing with two cysts and as the patient would not allow both sides to be done at the same time, that of the right side was operated upon.

The operative findings showed the cyst to lie posteriorly to the teeth and without connection with them, for at no time were the apices of the right incisor teeth seen. A few weeks later the left side was operated upon, but after breaking through the buccal plate high above the apices of the teeth, no cyst could be found. Evidently there was a single cyst and not a double one as the X-ray indicated.

In my experience at various clinics, I have observed numerous cases of X-ray of the teeth of upper incisor region, showing a small

circumscribed cyst-like area of decalcification in the median line of the maxilla. These areas were considered to be an enlarged incisive foramen, especially if the teeth in the neighborhood were negative from a dental standpoint.

Recently, while in Vienna at Dr. Gottlieb's laboratory, a maxilla of a young adult, who had just died, was brought in. Before decalcifying the jaw, it was suggested that an X-ray be first taken. The X-ray revealed a round circumscribed area in the median line between the teeth, similar to many others seen and which was always consid-

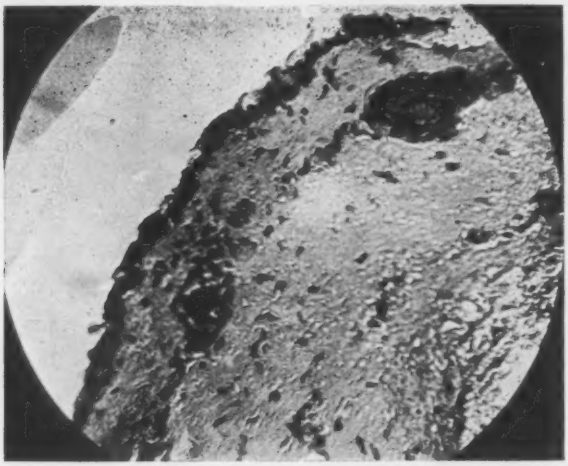

Fig. 6. Photomicrograph of the cyst wall of Case 2. (a) Epithelial layer consists of two rows of epithelial cells (ciliated and goblet cells).

ered to be an enlarged incisiva foramen. When the jaw was sectioned through this area and examined microscopically, it revealed that the so-called incisiva foramen was really a cyst that had no connection with the teeth (Dr. Kornfeld[5]). In 1926 Hans Wermuth[6] had reported a similar case from the jaw of a young adult and had considered it to be a cyst of the naso-palatine duct. Wermuth's studies also showed that in dogs and rabbits there was a continuous communication between the oral and nasal cavities through the naso-palatine duct.

THE NASO-PALATINE CANAL.

Approximately 2 c.m. dorsal to the inner margin of the nostril and in juxtaposition to the nasal septum, each nasal fossa presents a slight

depression in its floor. This depression leads into a small canal lined with mucosa, continuous with that which lines the interior nasal meatus. The funnel-shaped tube of mucous membrane, the naso-palatine canal (canal incisvus) courses obliquely caudad and with its fellow of the opposite fossae converges toward the nasal septum, descends almost vertically and passes through the Y-shaped incisiva foramen (anterior palatine canal) in the hard palate. The canal ends on the roof of the mouth at the side of the palatine papilla.

THE GENESIS OF NASO-PALATINE DUCT CYST.

With the fusions of the palatine processes of both maxillae, the oral cavity is divided from the nasal cavity. However, a wide communication between these cavities is still present in the early periods of fetal life anterior to the fusion through the naso-palatine ducts (Peter and Rydzeks[7]). In the vast majority of cases, the lumina of the ducts are later obliterated and are represented by impervious cords of epithelial cells continuous with the epithelium of the roof of the mouth at one extremity, and the funnel-shaped, ciliated, epithelial-lined depressions in the floor of the nasal forsae at the other. Occasionally in adults, the nasal palatine duct remains open and leads to a continuous direct communication between the nasal forsae and the mouth cavity. Leboucq[8], who used the ordinary thin probe in his work in 1881, first called attention to this finding. Later Rawengel[9], the disciple of Peter, studied the relationship of the naso-palatine duct in the new-born and adult by making serial sections of this area and examining them under the microscope. He studied 19 new-born cases and eight adults, and his findings showed:

	No. of Cases (27)	New-Born (19)	Adult (8)
Continuous passage	5	4	1
No passage	3	0	3
Blind sac from nasal side	14	10	4
Blind sac from mouth	4	1	3

His embryological studies also showed that there are three sets of epithelial tissues in this region: *1.* The naso-palatine duct and its rests; *2.* a middle epithelial cord in the common incisivus canal; and *3.* epithelial cells in the most posterior portion of the papillae. All the epithelial rests, aside from the naso-palatine duct, disappear in most cases in the first year of life.

Persistence of the lumina of portions of the naso-palatine duct may explain the epithelial cells in the maxilla dorsal to the upper incisor teeth. As Rawengel demonstrated, a blind sac either from the nasal or buccal cavity may exist, or in the event that both extremities of the duct become impervious, the central portion would be without a drainage channel and could readily become cystic.[10]

CYST OF THE FACIAL CLEFT AREA (FISSURAL CYST).

Case 2: A colored woman came to the Vanderbilt Clinic, Nose and Throat Department, complaining of a slight swelling along the left nostril and upper lip. As the nasal findings were negative, she was referred to the Oral Surgery Department.

Her history was that her friends first called her attention to this swelling, which would disappear and reappear. No pain or disturbance of function was present at any time. On inspection, a slight swelling at the left ala nasi region, which obliterated the ala nasi fold and slightly raised the lip, was noticed. On palpation, a definite small soft tumor was felt over the lip and side of the nose. The tumor was more noticeable in the vestibule of the mouth and was located over the lateral bicuspid areas of the alveolar process of the maxilla. No teeth were present in the maxilla. The tumor was soft and fluctuating (elastic), without signs of inflammation, tenderness or pain. Preoperative diagnosis was a root cyst that had eroded through the buccal plate of the alveolar process of the maxilla.

X-ray Findings of Area: No unerupted teeth or root; no area of decalcification. Eyes, nose, etc., normal.

Operative Findings: After the mucous membrane was elevated, a definite distended cyst membrane was revealed lying upon the alveolar process. The cyst membrane was easily freed on the sides and posteriorly, but it was held at the upper and lower poles. On manipulating the cyst, a definite tug in the vestibule of the nose, near the ala, became visible. The lower pole was then freed, but the upper attachment held firm, so that before it could be removed, some of the nasal mucous membrane had to be excised with it. A shallow depression at the junction of the root of the ala nasi and maxilla was found. The contents of the cyst was a thick, whitish, glary mucous fluid. Diagnosis after operation was retention cyst.

Microscopic Findings: The wall consists of an epithelial and connective tissue layer. The epithelial layer is very thin and consists of two rows of cylindrical cells, with a few goblet cells. The connective tissue layer is thicker. No glands or ducts present. No round cell infiltration.

THE GENESIS OF CYSTS OF THE FACIAL CLEFT AREA.

Over 60 cases found in this region have been published. The genesis of these cysts has been a subject of much speculation, research and controversy. The first case to be published was one by Zuckerkandl[11] in 1882, who, like many others that followed, simply described the cyst and its site. Most of the earlier writers considered them as retention cysts (Chatelier and Dunn[12]), Milligan[13] in 1894

and Knapp[14] in 1895 described a seromucoid cyst beneath the wing of the nose and collected six others from the literature. McBride described two others. Brown-Kelly[15] in 1898 described three cases and studied 12 others, whose age ranged from 19 to 58 years. While the cause was still undecided, the localization of all these cysts and the fact that they were not connected with the teeth was well established. Although B. Kelly postulated that these cases were retention cysts brought about through inflammatory processes of glands in that region, he called attention to the fact that "a possible embryological origin should, however, be kept in view, for the union of the superior maxillary and median nasal processes is effected or very close to the region in which the cysts develop".

Wingrave[17] and later Grünwald[18] in describing their cases mentioned the developmental possibility that they may have some connection with the naso-palatine duct. Kofler[19] had eight cases in all, one patient having a cyst in either side of the nose, that on the right starting 10 years before and causing an asymmetry of the face. The cyst stretched between the bone and mucous membrane of the aveolar process and nasal mucous membrane. A depression in the bone was also noted. In spite of its bilateral nature, the possibility of a congenital origin was not considered by Kofler, who also considered them retention cysts. Malon[20], Vogel[21], and especially Bruggemann[22], considered them related to the nose-lacrimal duct. Blumenthal, and especially Klestadt[23], however, maintained that these cysts were derived from epithelial cells of the facial cleft regions.

EMBRYONIC DEVELOPMENT OF THIS REGION.

The true genesis of these cysts is founded upon the embryological studies of this region by Peter[6], Kallius[24] and Klestadt. Actual facial clefts do not occur in human embryos, but rather slight furrows or depressions. Peter showed that the anlage of the nose is first apparent in the human embryo at about the third week as two thickenings of the ectoderm, one on each side of the naso-frontal process. These thickenings (olfactory placodes) soon become depressed below the surface and form the nasal pits or fossae. From the naso-frontal process, two secondary processes develop on each side, the medial nasal process and the lateral nasal process. Between these two processes is the depression, the nasal pit, which is the entrance to the future nasal cavity. As development proceeds, the two median nasal processes come closer together to form the single median process which gives rise to the median portion of the upper lip and a part of the nasal septum. The lateral nasal process form the lateral nasal wall and the ala of the nose. With the fusion of the

upper jaw and the middle and lateral nasal process, the naso-optic furrow is obliterated.

The fusion between the middle nasal process and the upper jaw occurs first, and later that between the middle and lateral process occurs. At the three points of fusion there remains at first an epithelial ledge which connects with the external ectoderm. Later this epithelial lamina is perforated by the mesoderm. Kallius was able to demonstrate the presence of epithelial cell rests in the mesenchyme for a long time after the second month. Peter also considered it possible to have dormant epithlial cells in the connective tissue from which growths might occur. Of particular importance for the histological picture of these cysts is the fact that these epithelial cell rests have the ability of forming respiratory epithelial cells.

Klestadt's studies of 34 human embryos demonstrates that these cysts were not connected with the anterior portion of the tear duct. His serial sections showed that the lacrimal duct never reached to the vestibule of the nose or the floor of the nose or ran laterally to the root of the ala nasi, the characteristic site of these cysts. Klestadt also contended that the possibility of these cysts arising from the naso-palatine duct was remote, inasmuch as they would involve the vestibule of the nose only secondarily and, as he said, naso-palatine cysts are very rare. Also, naso-palatine duct cysts occur near the septum. As a result of his studies, he considered them to be cysts derived from the epithelium of the facial cleft area (Gesichtsspalten zystem). Only on that basis could they be explained genetically.

Brüggemann's contention that they might arise from the most anterior portion of the lacrimal duct is based on Tüffer's work with mammals. Tüffer showed that sometimes there were two orifices in the nose, one in front and one in back, where the lacrimal duct came into contact with the mucous membrane of the nose. Tüffer, however, is of the opinion that the anterior orifice is phylogenetically the oldest and that this is shifted backward more in man, owing to the upward development of the nose.

Both Huizinga[25] (1925), who described three cases, and Doring[26] (1928), who described one case, agree that the Klestadt theory of fissural or facial cleft cyst genesis is the more likely and plausible one.

The characteristic features of all the cases described are: *1.* The typical situation of their occurrence, *i. e.*, at the junction of the root of the ala nasi, the vestibule of the nose, mouth and cheek. Also, that they have no connection with the dental cysts. This site coincides with the fusion point of the three processes. *2.* The cases microscopically studied showed this common feature—that the cyst

wall consisted of a thin epithelial and a thicker connective tissue layer. The epithelial layer consisted of single or double rows of columnar cells with cilia. Goblet cells were noted frequently. Glands and ducts were missing. The cyst contents varied from serous, seromucus to pure mucus; cholestern was negative. *3.* Of the 60 or more cases studied, only three occurred in males.

The contention that these cysts might be retention cysts is conceivable according to Klestadt, but, as he says, that does not explain their genesis. Since glands are found all over the nasal mucosa, it is inconsistent to believe that retention cysts should form only at this site. Also the presence of high ciliated epithelium would be improbable in retention cysts of nasal mucous glands.

The case presented tonight, while differing from the typical picture described, in that it was not visible in the nose, still had the typical location. The microscopic picture also conforms to that described. Hense it is presented as a fissural cyst, rather than as a retention cyst.

SUMMARY.

1. The usual classification of cysts of the jaws consist of *a.* root, *b.* follicular, *c.* multilocular or adamantinoma polycysticum. These are derived from the epithelium cells of the dental anlage.

2. Epithelial strands or cells rests from the naso-palatine duct in the foramen incisiva may occur and give rise to cysts in the median line of the maxilla, which may simulate root or follicular cysts in that region (Case 1).

3. Cysts from epithelial cell rests in the region of the fusion of the upper jaw, lateral and middle nasal process may occur. These cysts are situated partly in the vestibule of the mouth, nose and cheek. Offhand, they may simulate dental cysts. However, they are distinguished by being *on* bone and not *in* bone (Case 2).

4. The usual classification of cysts of the maxilla should be amplified to include these possibilities.

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267 West 81st Street.

CARCINOMA OF THE ORBIT AND ETHMOID REMOVED BY SURGICAL DIATHERMY.*

DR. M. C. MYERSON, New York.

This patient is presented to show what we can hope to accomplish with surgical diathermy in extensive invasion of structures about the head by cancer. Barnes has demonstrated the feasibility of attacking extensive malignant disease of the accessory sinuses and it is the aim of the writer to interest and stimulate those who come in contact with such cases to attempt to cure them, rather than consign them to the uncertain results that radium has to offer, or continued suffering and death.

This patient, a man age 58 years, had his right lower lid operated upon for lupus 22 years ago. A little over a year ago he presented himself at a hospital because of blindness of the right eye, pain in and around the orbit, and a tumor formation in the region of the inner canthus, which seemed to extend into the orbit. A section of tissue was taken from this area and a diagnosis of epithelioma was made.

The patient was first seen on the Eye Service at the King's County Hospital in May, 1928. At that time his right eye had not been functioning for over a year and the contents of the orbit presented a distorted mass. He was suffering excruciating pain in and around the eye. He had received five radium treatments before the time of his admission. He also complained of a stuffiness of the right side of the nose.

Examination of the nose disclosed a septum deviated to the right and a mucopurulent discharge coming from the middle meatus. X-ray study revealed an opacity of the right ethmoid region and evidence of chronic disease of the maxillary sinus of the same side. One could not differentiate between a long-standing suppurative ethmoiditis or a malignancy of moderate extent.

Dr. Moehle, of the Eye Service, was kind enough to remove as much of the orbital content as he could surgically. Following this, I applied the diathermic electrode to the remaining structures of the orbit and followed the infiltrative process as far as it went. The neoplasm took me into the ethmoid region, where by a process of

*Patient shown before Laryngological Section, New York Academy of Medicine, Oct. 24, 1928.

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coagulation the entire ethmoid labyrinth was exenterated. The sphenoidal cavity was also entered. There was no difficulty in penetrating the thin bone of the ethmoidal septa with the electrode. After the slough came away and the wound healed it was found that the entire middle turbinate had been retained and the extent of the sphenoethmoid operation could be made out. It was found also that part of the roof of the antrum had been removed, although this was not known until some small sequestra had been removed which left a space in the roof of the antrum.

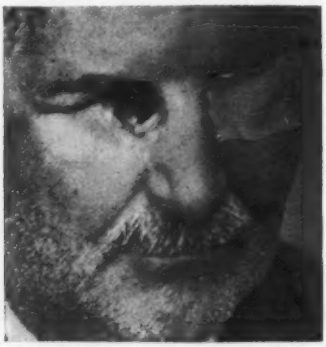

Fig. 1. Photograph showing extensive removal of orbital contents and extentration of ethmoidal region.

One cannot tell just how much or how little tissue he will remove in this region with the diathermic electrode, for there is destruction of bone, which does not make itself known to the operator until weeks have passed. For this reason the cribriform plate should be respected more during an electrothermic coagulation than during the usual surgery of the ethmoid and sphenoid.

The result is thus far satisfactory. The patient lost his headaches immediately after his reaction from the operation. There has been no evidence of recurrence since.

30 East 40th Street.

FRONTAL SINUS SURGERY, WITH CASE REPORTS.*

MAJOR ROBERT E. PARRISH, M.C., U.S.A.

In this paper I will divide the subject into surgical treatment of frontal sinusitis and surgical treatment of traumatic involvement of the frontal sinuses.

Frontal sinusitis is further divided into acute and chronic conditions, and cases with acute exacerbations are grouped as chronic.

Acute frontal sinusitis will nearly always respond to local treatment and unless complications result should never be operated.

Chronic frontal sinusitis requires operative treatment in a certain percentage of cases, but to determine this often requires a long preliminary period of observation and treatment. The first thing that should be considered in a given case of chronic frontal sinusitis is, what may be the relation of the other sinuses to the frontal sinus, and what can be done to clear up the disease in these other sinuses, and what abnormal conditions exist in the nose that may be of importance in the causation or exaggeration of the frontal sinusitis; such as deviation of septum; hypertrophy of turbinates; nasal polypi.

Unless some complication has developed which forces immediate operation on a frontal sinus case, I always begin by operating on the maxillary sinuses if they are involved, then on the ethmoids, if necessary. A submucous resection is nearly always indicated if the septum does not deviate away from the involved sinus. The next step is to remove the anterior tip of the middle turbinate, and in some cases the agger nasi. A period of time should be given between each step to determine if the frontal sinus symptoms will not clear up or improve sufficiently to preclude the necessity for further surgery. If the symptoms persist it is now time to decide whether it is worth while to do an intranasal operation on the frontal sinuses or to do the more radical external operation. A certain number of cases will be improved by intranasal operation, but in chronic cases, which type I am referring to, very few will be cured, particularly if the disease has affected the bone or the lining membrane of the sinus has become polypoid or developed into a pyogenic membrane. Further, there is a certain amount of danger when working through the nose, which far surpasses the danger of a well selected external operation.

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*Read before the Texas Ophthalmo-Oto-Laryngological Society, Dec. 10, 1927.

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External operations on the frontal sinuses should not be done except in well selected cases, when the indications are sufficient and after the proper "lead up" treatment. The "lead up" treatment, which I consider absolutely necessary before we should attempt to do a radical external frontal sinus operation, except of course in the emergency cases where complications force immediate operation. I will list as follows:

1. Careful X-ray study to determine the extent of accessory sinus involvement and size of the frontal sinuses.

2. Dental examination to determine if an ascending dental infection is present.

3. Correct nasal obstruction, deviation of the septum, and enlarged turbinates. A submucous resection is nearly always required.

Case 1. Large osteoma, left frontal sinus.

4. Operate the maxillary sinuses if this is indicated.

5. Open the anterior ethmoids if they are chronically diseased.

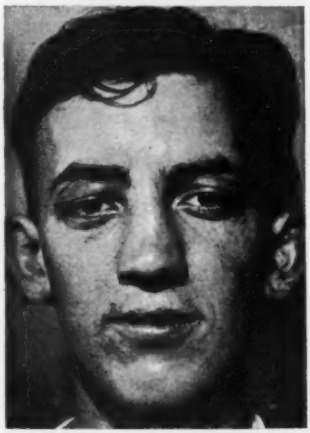
6. Remove the anterior tip of the middle turbinate.

7. Remove nasal polypi if present.

8. In some cases the indications now are such that we are justified in enlarging the nasofrontal duct.

9. During this entire period of treatment, which is carried out step by step with a suitable period of time between each step, local treatment of the nasal cavity is kept up. This, I will not discern, other than to condemn the use of nasal irrigation with watery solutions in these chronic sinus conditions.

When the afore-mentioned requirements have been carried out, and sometimes even earlier, the frontal sinus symptoms improve to such an extent that radical frontal sinus surgery is not necessary. But, if it is now necessary to operate you can expect to get maximum results from the radical operation in a patient thus prepared. Each step in this "lead up" treatment has improved the patient's condition sufficiently to amply justify it. Certainly no one can say that any unnecessary work has been done when we are working up the ultimate goal—a radical cure of a chronic frontal sinusitis. The choice type of external operation is a matter somewhat of a personal equation. Personally, the "Killian" operation forms the basis for the operation that I perform. Others get good results with the method



Case 2. Shows scar below brow, outer angle.

of Lothrop, etc. Ballenger gives the following objections to the "Killian" operation: First, the danger of permanent diplopia, due to detachment of the pulley of the superior oblique muscles of the eye; second, great deformity, in spite of partially removing the orbital roof, where the sinus is very large; third, the possibility that recovery may be obtained by less formidable operations.

DISCUSSION OF THESE OBJECTIONS.

1. The danger of permanent diplopia, when the pulley is detached, must be very slight—I have never gotten it, and my patients, with two exceptions, have never mentioned diplopia. If the pulley is detached, don't let them see with both eyes for 10 days. In dressing the

case, cover the normal side when the eye is exposed on the operated side. One of the exceptions mentioned was not handled properly and noticed diplopia for a few days following the operation. The other case noticed diplopia, so he said, for "a month or more". When the sinus is small I am often able to perfom the operation without disturbing the pulley.

2. The "great deformity", given as an objection, is well taken and there will be a good bit of deformity if too much of the outer table is removed, either above or below the superorbital ridge; but, removal of the diseased membrane, plus a large and permanent opening into the nose, is necessary to cure these cases, and there will rarely be

Case 11. Showing depression due to soft tissues of forehead dropping down and coming into contact with dura.

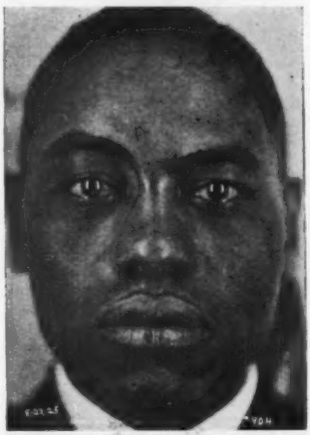
much deformity if care is taken to preserve the ridge and remove only enough bone to permit proper curettement of the sinus and to enable us to make a proper opening into the nasal cavity.

3. The possibility that recovery may be obtained by less "formidable operations" is not an objection to that operation, but simply means that sufficient care has not been taken in the "lead up" treatment and proper selection of operative procedures.

The following case history is a typical case where the external operation is indicated:

R. N., civilian, Veterans' Bureau. In May, 1918, began to have discharge from right side of nose; was treated at Naval Hospital,

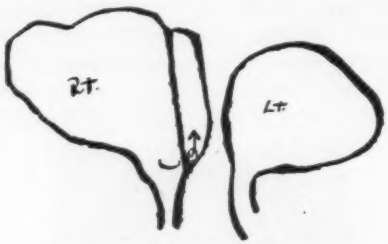
New York. Discharged from the Navy, January ,1919; continued to have symptoms but did not get any treatment for one year. March, 1920, went to hospital in Dallas, Texas; received local nasal treatments for 30 days, was discharged in August, 1920. Went to Public Health Hospital in Houston, Texas, for 60 days, had an intranasal operation on the turbinates and ethmoids, was discharged. In 1921, went back to Public Health Hospital in Houston, Texas, for 30 days; an intranasal operation was done on the frontal sinus; the sinus was then irrigated daily. In May, 1922, went to Naval Hospital, Washington, D. C., for 90 days; had nasal irrigations. In August, 1923, received local treatments daily for 30 days by a specialist in Dallas, Texas. Went to Veterans' Bureau Hospital at Muskogee, Oklahoma,



Case 3. Slight elevation of brow.

in September, 1923, and was held there until March, 1924; had nasal irrigations. In November, 1924, went back to Muskogee for 30 days. In the meantime, he had had another opening made in the frontal sinus by a specialist in Oklahoma City. February, 1925, came to this hospital; had argyrol tampons; was here 30 days. In 1926, went to Army and Navy Hospital, Hot Springs, Arkansas, had an intranasal maxsinusotomy; was there 30 days; went back to Muskogee, May, 1927; local treatment for 30 days; came to this hospital, Aug. 26, 1928; had a radial frontal sinusotomy on Sept. 15—was well in 10 days. Prior to the operation at this hospital, he was tender over the frontal sinus and said that the pain had been almost constant for

the past three years; no pain since operation. The sinus was not very large, the pulley was not detached, and the sinus was filled with pus and pyogenic membranes. I don't believe any procedure other than the radial operation would have cured this case. The history speaks for itself. The patient had spent 18 months in hospitals for this condition since 1918.



Case 4. Drawing to show compartment, right frontal sinus.

Case 4. After recovery.

When the indications for an external operation are met there should be no hesitancy in recommending it, as the results to be expected are such as to make it our most valuable procedure. It will not only cure your patient, but it will prevent dangerous complications in a high percentage of cases.

The operation which I have performed for the radical cure of chronic frontal sinusitis has served its purpose in such a satisfactory manner that I will give a brief description of it. While not original, this technique is assembled from numerous sources. The "Killian" incision is made, together with the method of Killian, of leaving a covering of periosteum of what will later become the bridge. The length of the incision should vary with the size of the sinus. As a matter of fact, some of the underlying tissues are often left attached to the periosteum, I am so anxious to leave it intact. An opening is made into the frontal sinus above the ridge, sufficient in size to permit me to curette out all diseased tissue by means of the Coakley set of

Case 5. The outer table of the right frontal bone was eroded through, with free pus under scalp at time of operation.

curettes. The orbital tissues are now stripped back, the pulley may or may not have to be detached, depending on the individual case, size of sinuses, supraorbital ethmoid cells, etc. An opening is now made through the floor of the sinus, beneath the ridge and down on to the nasal bone. The duct is now probed for size and direction. A rasp is placed into the duct and by rasping, away from the vault, a large opening is soon made into the nasal cavity. This is done from above downward. This opening should be sufficiently large to permit the placing of a rubber tube the size of a lead pencil, or larger, and also a strip of iodoform gauze both from above downward, extending through the nose. The tube is fenestrated and the upper end is placed

in the sinus cavity. The wound is closed, the periosteum being brought down over the opening above the ridge. A rubber tissue drain is placed at the angle extending down, above and behind the orbital tissues. Dermol sutures close the wound up to the eyebrow, and black silk is used in the brow. The brow should not be shaved. If the pulley is cut it can reattach itself to the periosteum covering the bridge. The gauze is removed in three or four days, the rubber tube is left in 10 days, the rubber tissue drain is taken out in 24 hours, the dermal sutures in three days, the black silk in five days. The nose is not irrigated. Any discharge present is wiped out with dry cotton. The tube can be kept open by the use of dry cotton on an applicator passed up and down it by a pump-like action.

Case 6. Before operation.

In performing the operation the periosteum covering the frontal bone should be disturbed as little as possible. Strip it back just far enough to remove the required amount of bone. The nutrient blood supply of the frontal bone is derived from the vessels in the periosteum and from the vessels of the dura. The field of the operation being infected, the blood supply is essential to supply resistance. The frontal bone being a "flat bone", has little resistance when infected. osteomyelitis once set up tends to spread, being limited only upon reaching the suture lines. Osteomyelitis of the frontal bone is usually secondary to sinusitis, syphilis or trauma.

I believe in using general anesthesia, ether-vapor, the posterior nares being packed. An airway is placed in the mouth to prevent the

tongue and soft palate from interfering with respiration. A perforated airway is best, when a hook is used for the ether-vapor. Suction is also a great aid in keeping the field of operation clean.

This operation cures frontal sinusitis and my patients have always been pleased with the results obtained. The scarring is not considered sufficient for patients to particularly fear the operation, when the operation is done as previously described.

Certain traumatic injuries to the frontal sinuses may require operative interference and the selection of the proper type of operation is of equal or more importance than the selection of the operation for the cure of frontal sinusitis. When there is a compound fracture of the outer table, with loss of substance, care should be taken in removing the fragments and the sinuses should be cleaned out, as in a radi-

Case 6. After operation.

cal operation. Overhanging or sharp projecting bone should be removed and the wound should be permitted to granulate up, with external drainage, if there has been injury to the posterior table. The sinus should not be connected with the nose for fear of getting virulent organisms into the sinus from below if there is reason to believe that the posterior table has been fractured. If the dura is exposed the external soft tissues should be allowed to drop down and come in contact with it. This will tend to prevent infection through the dead space in the sinus cavity, the infection most likely arising in the nose. Another writer drew attention to this point in a recent article in one of the journals, but I am unable to give his name at this time.

When there is no depression or injury to the dura and no external communication with the sinus, there will be no need for surgery, unless to replace a depressed fracture of the bridge.

DESCRIPTION, CASES, PHOTOGRAPHS.

Case 1: H. J., colored, age 28 years. This patient had an X-ray taken of the sinuses, because of some acute infection and a condition which was probably an osteoma was noticed in the films. The acute symptoms cleared up and the patient was watched for about two years, during which time he was free from symptoms and there was no apparent change in the size of the opacities. Osteomas of the frontal sinuses are not rare, may cause pain, but often produce no

Case 8: Wound not completely healed when picture was taken.

symptoms; unless symptoms are being produced it is probably not necessary to disturb them.

Case 2: A. S., white, age 22 years. This patient shaved his eyebrow prior to the operation; he thought it would be necessary. You will notice that the incision runs low at the outer portion, because of the difficulty in following the brow when hairs are removed. This is the only time I have ever operated on a shaved brow.

Case 3: D. W., colored, age 27 years. This case shows just as an example scarring resulting from this type of operation.

Case 4: T. N., white, age 25 years. This is the only case that I have ever had to reoperate on who I have perforemd the primary

operation. This case had a peculiarly-shaped frontal sinus, which did not seem so apparent until the condition was studied very closely (see drawings). About 10 days after the first operation there was still considerable mucoid discharge coming out near the angle of the eye and I decided that the sinus should be reoperated. This was done, and a septum forming a compartment was found that communicated with the sinus at its lower portion. The septum was removed, and the compartment cleaned out. The case then healed by first intention.

Case 5: H. W., white, age 23 years. This patient had a chronic maxillary and chronic frontal sinusitis, right side. A radical Caldwell-Luc operation was done. An intranasal and partial middle turbinotomy was also done. Patient insisted on going to duty, as he was an officer in charge of a finance school, which was then in session. He was being treated daily in an ear, nose and throat clinic in the city, but after a few months developed more acute symptoms, with tenderness and swelling above the right eye. A frontal sinusotomy was performed; the incision made through the scalp and an opening found through the anterior table, with a collection of pus underneath the scalp. The usual operation was done and recovery was rapid and complete.

Case 6: W. H., white, age 24 years. Patient came to hospital with marked exophthalmos and external deviation of the right eye. There was a discharging fistula region, inner canthus. An intranasal ethmoidectomy was done, first in one week a radical maxillary sinusotomy (Caldwell-Luc), and in about three weeks the radical frontal sinusotomy was done. Recovery was complete.

Case 7: L. B., white, age 26 years. A case of bilateral, frontal sinusitis, chronic, suppurative, originally a pansinusitis. The antrum and ethmoids had been previously operated. Patient developed erysipelas about 10 days following right radical frontal sinusotomy, which was followed by bilateral extensive osteomyelitis of the frontal bones. Meningitis developed and patient died after about six weeks. At autopsy both frontal bones looked like a piece of worm-eaten wood. The left frontal sinus was operated when it was evident that there was a communication with the right side and the infection had involved the tissues about the left orbit. This, however, was not done until after the most acute symptoms of erysipelas had subsided.

Case 8: G. L., white, age 29 years. A case requiring a reoperation on the frontal sinus. A previous radical operation had been done by a specialist in Washington, D. C., but it was evident when the sinus was reopened that his failure was due to insufficient opening being made into the nose. Also, he had failed to remove all the mucous

membrane at the upper inner angle. Patient made complete recovery and I don't believe he will have any more trouble. This photo was made before the wound had quite healed, the area showing black is where a rather wide scar from the preceding operation had been cut away. I have reoperated numerous cases and in each case the opening into the nose either had not been made at all, or was not of sufficient size to drain the sinus cavity.

Case 9: H. T., white, age 26 years. One year before patient came under care of writer, he had a severe attack of acute sinusitis with rupture into the orbit. A Lynch operation was done by a competent specialist in San Antonio. Patient left care of the surgeon before recovery and developed fistula above eye and temporal region. Continued in this condition with practically no treatment for nearly one year. Came under my care with extensive osteomyelitis, left frontal bone, with considerable loss of substance through expulsion of sequestrae with six discharging fistulae around suture line, frontal bone vertex to external orbital ridge. Radical operation done as usual, except no attempt made to open into nose. Nasal duct packed off. Two months later a second operation was done, the scalp turned down and entire suture line of frontal bone exposed. All dead bone removed, dura exposed for more than 4 inches, junction frontal with parietal bone. Flap held back and wound Dakinized. After all signs, suppuration gone, and granulation complete, a third operation done to replace flap of scalp. Union was complete without any further suppuration of frontal bone, no more fistulae developed.

At the first operation on the frontal sinus, numerous sequestrae removed from roof of orbit. Discharge from sinus gradually diminished. Patient left hospital against advice and had no further treatment for about eight months, when he returned on account of a leg injury. There were no recurrences of the osteomyelitis of the frontal bone above, and there was almost no sign of sinus infection. There was a very small fistulae with occasional moisture above inner canthus. Considering initial condition, no further surgery was advised. It might be said that patient made a clinical recovery.

Case 10: J. B., white, age 24 years. This patient was shot with a .22 rifle, the bullet passing through the left frontal sinus. He came under my care six months later, with a foul, discharging fistula at the inner angle of the eye. This fistula communicated with the frontal sinus. The X-ray examination showed that the sinus was filled with fluid or membrane and showed the bullet just inside the skull, with a fragment in the left side of the brain.

A two-stage operation was decided upon—first to clean out the sinus and remove the bullet, if it could be reached external to the dura, with external drainage, and secondly, to reopen and communicate the sinus cavity with the nose, as is done in any other radical frontal sinus operation—this to prevent the possible infection of the cavity by virulent bacteria from the nose, which is more probable if only a one-stage operation is done.

The first operation was done, the badly suppurating sinus cleaned out, fragments of the inner table were removed, but the bullet could not be reached without opening the dura—this was not done. The condition improved rapidly and after about six weeks the second operation was done. The wound had closed, the stitches were removed, the packing was out of the nose, the patient was up and about, ready to go home, when he was suddenly taken ill, developed acute streptococcic meningitis and died. This was about eight months after the gunshot wound. Unfortunately, the dead space in the sinus was not obliterated and nose allowed to communicate with the sinus. A faulty procedure.

Case 11: A. M., white, age 29 years. This patient was hit by a baseball bat, the bat leaving the hands of the batter, with evidently great force. The outer table was knocked away, a wide opening made in the soft parts, the posterior table fractured and three pieces displaced inward. Patient was seen and the wound opened up in two hours after the injury; the sinus cleaned of blood and debris and the three fragments of inner table removed. The soft tissues were let down to come in contact with the dura; the lower part of the sinus was packed off to prevent infection from the nose reaching the dura, and the wound was let close. The wound healed; he has had no further trouble.

Beaumont General Hospital, El Paso, Tex.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Regular Meeting, Tuesday, Dec. 4, 1928.

Blood Conditions of Interest to the Otolaryngologist. Dr. Edward Steinfield.

I have divided my topic tonight into two parts: 1. The discussion of the use and interpretation of blood examinations prior to operations upon the nose and throat as a precaution to detect tendency to hemorrhage. 2. A brief review of some diseases of the hematopoietic system which are apt to be seen by the otolaryngologist when epistaxis or other hemorrhages occur from the upper respiratory passages.

Under the first heading come tests of the blood coagulation mechanism. Though the usual routine is finger puncture, this does not give the true coagulation time. Estimation of the coagulation time of a small amount of blood removed by vein puncture and expelled in a small test tube represents more nearly the true coagulation time. This procedure is of value in detecting hemophilia but it is necessary to do bleeding time and other tests to detect hemorrhagic purpura (essential thrombocytopenia). These other procedures are blood-platelet counts, clot retraction and tourniquet tests.

Though the presence of moderate anemia does not necessarily interfere with plans for tonsillectomy and other operations, I believe caution should be used if the leukocyte count is low. There is in these cases a distinct danger of necrotic lesions at the site of operation. This type of lesions is noted spontaneously in many aplastic states when leucopenia occurs.

It is important to discuss diseases of the hematopoietic system because of their possible presence when contemplating operation and also in diagnosis of epistaxis or other hemorrhages. I am not, of course, considering here cardiovascular disease. Hemophilia and hemorrhagic purpura I have mentioned. An interesting syndrome is familial epistaxis with telangietasis: nose bleed occurs in members of the same family and dilated capillaries are noted upon the face and nose. No abnormality of blood coagulation is noted. Hemorrhage occurs in aplastic anemia and leukemia. It is also necessary to consider the possibility of the presence of leukemia, Hodgkin's disease or lymphosarcoma in unusually marked hypertrophy of the tonsils.

DISCUSSION.

Dr. Wieder: With reference to bleeding time, I might say that it has always been more or less a puzzle to me. It seems that the bleeding time will vary, depending on the incision. I would like to know if an incision of different size will bleed accordingly. When you have a case in the hospital you get all kinds of reports from residents. I am glad to hear Dr. Steinfield bring out the difference in coagulation time in hemophilia and thrombopenic purpura. I have never had that called to my attention before and it explains why some of our girls bleed to death, and not the boys. I remember an operation done by an assistant on a brother and sister. The boy proved to be hemophilic and bled to death. The sister almost bled to death from apparently the same cause. I was never able to quite explain it, because I was always taught that you do not have hemophilia in boys. This is an important addition and I am very glad Dr. Steinfield called our attention to it.

Dr. Burns: I have been led to believe in my past experience with coagulation in veins and arteries that coagulation is entirely different in veins and arteries. I have made 17 tests on venous and arterial blood coagulation. In all these cases I found a difference of over two minutes. In veins the coagulation time is longer.

Dr. Steinfield (in closing): Dr. Wieder put his finger on the weak spot of the whole business. There are errors in all types of work, whether it is done by skilled or unskilled workers. If you take a normal individual and make a deep puncture, he will bleed longer than if you stick him lightly. The men

doing this work familiarize themselves with the test. The instrument should always be a sharp one. If you take an individual with hemophilia, puncture him and at the same time bruise him, he will bleed long. The type of instrument has a great deal to do with it. If you want to get the right coagulation time, the finger method is a fallacy. After all, a great deal of your work is done on small children and you would have a job getting into the veins. If you do do coagulation time with that method you can receive the blood on glass slides or you can take it in long drawn out capillary tubes. You can make it flow up 6 or 8 inches of capillary tube and all you have to do is break off an inch of the tube at a time. A miniature clot comes right out of it. The almost flawless method is the simplest one—the test tube method. The bleeding time is usually taken from the lobe of the ear. You have to get some history of the case before operation. In a physical examination you can detect various manifestations. If you do a fairly reliable bleeding time and coagulation time, you can spot the two main bleeding diseases. With all that, you are going to get some discrepancies.

It is impossible to have a great difference between venous and arterial blood in respect to coagulation. When operating you are dealing with blood of arteries, arterioles, venous sinuses, etc. When taking coagulation tests by the test tube method you are dealing with venous blood. In taking a test from the finger you are taking capillary blood. Capillary blood is the same as the blood in the arteries. If you take the blood from the arm there is a difference, due to the method. In my own experience I have never found any particular difference in coagulation. The only way of doing arterial tests is by going into the radial arteries.

I brought out that point at the beginning of my talk. As a matter of fact there was a difference of seven to eight minutes in the time. I thought you meant blood as it exists in the arteries. I misunderstood your question.

Ceanothus Americanus—A Study of Action on Coagulation Time in Normal Individuals. Dr. Anthony A. S. Giordano.

Dr. Giordano read a paper describing experiments with 62 medical students. Their coagulation time was tested and then a preparation of ceanothus Americanus ,was administered to them. Their coagulation time was noted at one-half hour, one hour and then one-and-a-half hours after the administration of the drug. The following were the conclusions:

1. The alkaloids of ceanothus Americanus definitely reduce the coagulation time in the normal person. Our observations show this to be a reduction of 20.5 per cent.
2. The maximum effect seems to be obtained within one-half hour and to last through a period of one hour, after which a return to normal comes on rapidly.
3. The dose of 2 drachms gave a maximal effect; a larger dose did not materially depress the coagulation time to a greater extent.
4. Therapeutically, therefore, it would seem that the best advantages to be gained are by giving a dose of 2 drachms and repeating this hourly in order to produce a continual maximal effect.
5. While preparations of ceanothus Americanus do have the effect claimed for them, no one should expect to prevent or check hemorrhages by the use of this drug alone. It may be used as a possibly helpful adjuvant, but not to the extent of neglecting accepted precautions and, when hemorrhages do occur, adopting prompt and energetic surgical procedures.

DISCUSSION.

Dr. Cohen: I have used ceanothus on a number of cases and my experience was that giving it before operating, it helps. After operation everyone would vomit. I should like to hear some suggestions for its practical use.

Dr. S. Skillern: I have been using ceanothus for about two years. As Dr. Cohen said, the best results are obtained by giving this drug before operation. This I have found especially true in radical maxillary sinus operations. In these operations, there is always a good deal of bleeding, followed by a post-

operative oozing. I use ½ oz. of ceanothus one hour before operation, another ½ oz. is given as soon as the patient is fully out from the anesthetic. This stops the oozing of blood. It is very annoying to a patient to have blood running down the back of the throat or over the lip and one is never sure just how much blood the patient is losing.

In regard to Dr. Cohen's question, possibly I can help out. I have given ceanothus in every way. I have given it in orange juice, lemon juice, water, etc. In my experience the best manner to give this drug is in a tumbler filled with cracked ice; in other words, frappe it. If this is followed by sodium bicarbonate, the patient will as a rule retain it. Sometimes in tonsil cases, ceanothus does make them vomit. I believe it is more the blood and ceanothus combined that nauseate them than the ceanothus itself, although some patients do not seem to be able to retain this drug no matter how it may be given.

DR. STEIN: I want to make the statement that I do not think anything can take the place of good surgery in operations about the nose and throat and ear.

I would like to report a case of bleeding in a mastoid operation which started two days after the operation and bled from various parts of the wound for a number of days. This patient was given ceanothus 2 drahms every hour for several days with no apparent effect on the bleeding. I applied firm pressure to the mastoid wound, which was the only thing that controlled the bleeding. The same thing applies to post-operative hemorrhage in tonsillectomy. Here, again, I believe that good surgery will prevent hemorrhage, but should hemorrhage occur, I do not believe anything will take the place of firm pressure or picking up the bleeding point and applying a suture. I, personally, depend more on these mechanical means of control rather than to attempt to change the coagulability of the blood. If bleeding persists, I invariably insert a sponge and sew up the fossa tightly. This has never failed to stop a nasty bleeding.

DR. WIEDER: In cases that require a coagulant I have been using calcium. It has helped to increase the coagulation time. However, I feel that the main factor that determines whether we have hemorrhage or not is the mechanical condition produced in the vessels at the time of operation. If the intima of the vessels is injured sufficiently, clotting quickly takes place in the vessel and you have no hemorrhage. If, however, the vessel is cut probably without injury to the intima, hemorrhage is likely to occur. It has been proven that vessels can be held closed for long periods of time by gentle pressure without intramural clotting. Therefore, especially in using force instruments, pressure must be made sufficiently strong to crush the intima, or if done quickly enough, to accomplish that same purpose would be sufficient without holding. However, the pressure should not be maintained long enough to cause death of the end of the vessel, otherwise we would have secondary slough and secondary bleeding. Almost any bleeding can be stopped if the intima is properly handled. Dr. Allis, a former chief, claimed that hip joint amputation had been performed without the use of a single ligature by simply twisting the bleeding point, including the femoral artery.

DR. STEINFIELD: Did you recommend ceanothus Americanus locally? It certainly merits trying it in order to shorten coagulation time. I presume trying ceanothus Americanus on an individual with hemophilia would be very instructive. I imagine the difference would be more appreciable than in normal individuals.

DR. GIORDANO: The only way I could possibly answer is that I have tasted the drug myself and it has a very pungent taste. It seems to pucker up the membranes. This is due to the alkaloid content. I believe it is the bitter taste. anesthetic and infected blood which cause patients to vomit. The tests referred to in my paper were done on normal individuals. Ceanothus Americanus does not cause vomiting *per se*. When it does occur, some outside cause is to be found, such as ether or infected blood, plus drug.

Dr. Wilbur used ceanothus Americanus in one case of lymphatic leukemia and found the coagulation time was lowered.

I do not know whether ceanothus Americanus would work locally, since it was not tried by us.

Regular Meeting, Wednesday, Jan. 9, 1929.

Case Presentation for Diagnosis. Dr. Arthur J. Wagers.

The case I wish to present for diagnosis is that of a man, age 63 years, who first came to the Clinic at Jefferson Hospital in July, 1928. His only complaint at that time (and since) was merely annoyance due to the presence of a very small area located in the roof of the mouth opposite the second molar tooth and external to the median line of the palate. The spot complained of has never been painful, but he is always feeling it with his tongue.

On examination, the entire roof of the mouth was found to be white in color, presenting very much the appearance of having been burned by the application of phenol. The small area which was disturbing to the patient could be seen, but differed from the surrounding area only in the fact that the surface was slightly rough and not as uniformly white as the surrounding tissue. It was neither elevated nor depressed. There was no sign of ulceration.

This man has enjoyed general good health all his life except for an attack of pneumonia 18 years ago. During the past two years he has been employed as janitor. Previously he had been employed in the building trade. He appears to be in good physical condition, is not losing weight, eats and sleeps well. He is a heavy smoker of the pipe.

The usual laboratory studies were made. Blood Wassermann was negative. Differential blood count approximately normal. Urine normal. Smear taken from roof of mouth showed absence of spirochetes. A few cocci and bacilli were found.

There appears to be nothing in the history or study of the case to enable us to make a definite diagnosis.

Treatment by means of various local applications over a period of six months has not altered the appearance of the palate.

I have brought this patient here in the hope that someone among you may have observed a similar case and could perhaps tell me if this condition is likely to prove the forerunner of a malignant change.

DISCUSSION.

Dr. R. H. Skillern: About a year ago a man came into the office while one of the students was assisting me. He had exactly the same appearance as Dr. Wagers' case. We made every test I can think of. The Wassermann test was negative, the tuberculin test was negative. We cultured for diphtheria and leptothrix. We prescribed some local treatment; gave him injections of serum, also various vaccines. None of these things did him any lasting good. The local applications were efficient for a while. The pigment that appeared most efficacious was an application of equal parts of tincture of iodine, tincture of benzoin compound and glycerite of tannin, equal parts of each. Under treatment with this mixture he got well. I think that man came over a period of six months and he dropped in two times a week for treatment.

Latent Mastoiditis with Necrosis. Case Report. Dr. George W. Mackenzie.

(To appear in a subsequent issue of The Laryngoscope.*)*

Dr. Wieder: I would like to ask Dr. Post if he considers the area in the right mastoid necrotic and does he think that it is remaining as a foreign body, surrounded by a bone, or does he think the bone has been totally absorbed? If it is considered necrotic, at the time of operation, why were both sides not operated on at once?

Dr. Post: Dr. Wieder, the area on the right side represents a focus of pathology which has been active at some previous time. At the present time, however, I believe it is inactive and is walled off and surrounded by sclerotic bone. I would not regard it as an operative mastoid from an X-ray aspect.

On the affected side we see distinct evidences of irregular resorption and necrosis extending upward with a well defined break in the line of the tegmen tympani. These findings, along with headache and other clinical symptoms, would serve as indications of an operative mastoid.

I would say that the mastoid on the right side is a weak link in the chain and would be subject to go the same pathological course as the one on the left side. From its general appearance on the roentgenogram, I would feel that cultures from it would be negative. It may, however, flare up at any time.

Case Reports: Nasopharyngeal Fibroma Treated by Operation and Radium. Dr. Abram H. Persky.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DR. ERSNER: I saw Dr. Coates' case in 1917 and it was very troublesome at that time. Every night I was called to stop bleeding. At that time I used a Lewis snare and we broke three or four wires before we were able to remove the growth. I recently saw the boy. He is 27 years old. He has large nares on the left side. He has not had a recurrence of the growth. The growth disappears after 25 years of age. I think the thing that impressed me every time I looked at my patient was, that I visualized the hemorrhages I had with Dr. Coates' case. It is peculiar that our three cases were male. They all started on the left side in the region of the sphenoid. After you remove these growths, the result of the pressure on the surrounding structures is so great that in a child not more than 13 or 14 years old, we are able to place two fingers in the nares. I thought at the time of removal that since we had to use post-nasal packing, that it may be a good thing to inlay radium emanation into our post-nasal packing. In our second packing the radium was placed in some gauze and placed into the nares. So far there was no recurrence.

I want to thank Dr. Persky for writing up these cases. There are often good cases that we run into but we do not have the time to write them up. Dr. Persky is to be congratulated on the way he wrote these cases up.

Case Report: Chronic Empyema of Maxillary Sinus, Complicated by Acute Mastoiditis of the Same Side. Dr. Oram R. Kline.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. ALEXANDER: I would like to know how Dr. Kline determined the absence of polypi through the opening of the nasal wall of the sinus.

DR. KLINE: We were able to look into the antrum but it was impossible to tell completely. The antrum we saw had no polypi present.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

December 17, 1928.

Dr. Herschel Ezell, Chairman, Presiding.

Atypical Mastoiditis. Dr. Herschel Ezell.

M. R., age 5 years, white, was referred to me by Dr. Owen Wilson on Dec. 5, 1928, complaining of swelling back of the left ear. There had been considerable pain. The mother stated that the child's ear had been swollen for two weeks and she had been sick with the "flu". She also stated that the ear discharged for three days two weeks ago, but there had been no discharge since that time.

Examination: The child carried her head tilted considerably towards the left side. The mastoid was greatly swollen and was quite tender. The mother said that the mastoid was not so badly swollen as it had been. On inspection a small perforation was found in the posterior wall of the canal, which was discharging a few drops of pus. The drum was normal, or perhaps slightly injected. The hearing was not as acute in the left ear as it was in the right ear.

Clinically, we had a picture of mastoiditis, but Dr. Wilson and I decided that it would be best to X-ray the patient before subjecting her to an operation. The patient was thereupon referred to Dr. H. S. Shoulders for roentgenographic study, who reported that there was no evidence of pathology from the X-ray pictures and that they were very much alike. It was hard to co-ordinate this report with the physical findings. Regardless of this, we decided not to operate but to keep the patient under observation and to treat her locally. The mother was instructed to irrigate the ear with very warm water four times daily. The external ear has been treated with alcohol and boric acid and the perforation has closed. Also the mastoid swelling has about subsided.

We are not sure that we have pursued the right course in this case, but under the circumstances we thought best not to use surgery. There may be a re-occurrence and it may yet be necessary to operate. So far the patient seems to be doing nicely.

DISCUSSION.

Dr. Fred Hasty: If I felt sure there was a fistula in the posterior canal wall I should operate. I have been too slow to operate on these cases. We should X-ray in acute stages, later, and again if necessary, but not let one X-ray change our opinion to a great extent. A mastoid operation well done is not considered dangerous.

Dr. Eugene Orr: I agree with the Doctor. I believe that we ought to be conservative in operating, but still I think we are too conservative. From what Dr. Ezzell said, I would think that I was jeopardizing the life of the patient if I did not operate. I don't think that we should depend too much on X-ray.

Dr. E. B. Cayce: I agree with what has been said about X-ray. No one thing can be taken to decide a case. All the findings should be considered. I think the man who said that he would not operate without an X-ray and that he would operate with a positive picture is wrong.

(Signed) W. W. Wilkerson, Jr., M.D., Secretary-Treasurer.

International Digest of Current Otolaryngology

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St. Louis Jewish Hospital E.N.T. Journal Club.

The editorial staff of this column wishes to voice its appreciation for the comment expressed concerning this new Digest of Current Otolaryngology. Readers are again invited to submit for publication any news of scientific otolaryngologic interest. The purpose of this column is to stimulate interest in scientific research and to keep our readers informed in a brief way of what is being done along these lines in various centers of the world. We invite brief preliminary reports of research or new ideas in this field with the understanding that such reports shall not prejudice subsequent complete publication. Enlightening group or society discussions on recent research or new methods of therapy, etc., are also invited.

The Twelfth Annual Meeting of the American Bronchoscopic Society will be held on Saturday, July 6, 1929, at San Francisco. The meeting place will be the Fairmont Hotel. Communications should be addressed to Dr. Louis H. Clerff, Secretary, 128 S. Tenth street, Philadelphia.

We are in recent recipt of a new journal called "Revista Cubana de Oftalmologiay Oto-Rino-Laryngologia", a monthly journal edited in Havana, Cuba, in Spanish, by Celestino R. Arguelles and Tomas R. Yanes with the aid of a distinguished Cuban collaborating staff. The scientific articles appear in rather more elegant form than we are accustomed to finding in our journals, the cover and paper are in keeping with marked artistic decorations prefacing and ending each article and each section. An interesting part of the journal is an analysis of recent medical literature. We wish the Revista Cubana de Oftalmologiay Oto-Rino-Laryngologia all success.

In view of the fact that Ephedrine is so commonly used in our speciality it is interesting to note that Higgins, of Richmond, reports in the Journal, American Medical Association, January 26, 1929, a case of chronic ephedrine poisoning. The case was a hyperthyroidism with frequent attacks of asthma. Ephedrine ½ grain

was taken after each meal. The symptoms were: Lassitude, nervousness, restlessness, aching over the body, frequent chills, weakness, insomnia and constant loss of weight. Within 24 hours after the discontinuance of ephedrine all symptoms were definitely lessened but insomnia, lack of appetite and emotionalism did not disappear for three weeks. It might be of interest to have further reports on this question.

H. S. Reichly, of Cleveland, reports a case of paroxysmal sneezing which turned out to be an early symptom of whooping cough. He adds that the paroxysmal coughing may be entirely absent and paroxysmal sneezing take its place. To Rhinologists this may prove to be a matter of some concern.

On his discussion of the relationship of diseased teeth, infected tonsils and sinuses to heart disease in children, Kerley, of New York, says: "If all children with diseased teeth, infected sinuses and infected tonsils developed heart trouble, few would escape. As it is, few of the great family of children develop this disease and these are the unfortunates whose bodies supply the favorable soil." He classifies a certain group of children as potential heart disease cases and it is in this class where he recommends immediate removal of the focus of infection. Coming from a large pediatric experience these words carry much weight.

Clinical Significance of Pain at Mastoid Tip in Acute Otitis Media Busacca, of Bologna, in the Arch. Ital. di Otol., rinol., e laringol., XXXIX Fasc. 7, 1928, says that this pain at the so-called "Caliceti's Point" (between the angle of the jaw and the mastoid tip), is no indication of what he earlier described as incipient mastoiditis. Two years of additional study of this question in Caliceti's Clinic has shown that persistent pain at this point is not necessarily indicative of a mastoid reaction, incipient or otherwise, but is always due to a middle ear disease, even in the absence of otoscopic findings, the pain being due to pressure upon the auricular branch of the vagus in the canaliculus mastoideus.

Khayel, of Padua, describes an interesting experimental study in the Arch. Ital. di Otol., rinol., e laringol., XXXIX Fasc. 7, 1928, on The Behavior of the Nasal Mucosa to Inert Powders. He subjected rabbits to inhalations of soot-laden air. In every instance the author

finds that the first inhalation and only the first, provoked an acute muco-purulent reaction. Excessive inhalations, even after a period of rest, provoked only moderate reactions hence the author believes that the nasal mucosa develops a tolerance to minimal traumatism with the inhaled air. He proved this by sectioning the mucosa of a rabbit killed while in a period of rest and the epithelial cells although literally filled with carbon were in a state of indifference showing hardly any reaction to the presence of the foreign substance. It is noteworthy that reactions were most intense in the maxillo-turbinate region of the nose rather than in the higher olfactory region. Histological examination revealed elongation of the epithelial cells and their interiors filled with carbon particles and enlargement of the goblet cells.

Where a marked reaction was present in the nose, the author was also able to observe a corresponding reaction in the epithelium of the trachea and larger bronchi but less pronounced and with less infiltration of carbon particles.

J. S. WRIGHT, of New York, in The American Journal of Diseases of Children, January, 1929, reports a peculiar case of bilateral gangrene of the feet and toes following tonsillectomy. The case followed a septic course but recovered with a residual cystolic murmur and the loss of one toe.

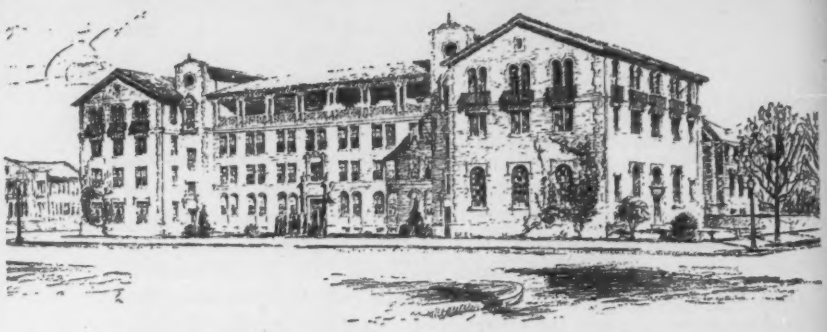
HALLE, of Berlin, in the Medizinische Klinik, December 21, 1928, reports good surgical and phonetic results in late operations for defects of the palate. Because of the vomiting following general anesthesia he prefers local anesthesia. He states that his cases show such good results that he now seriously questions the old maxim of "essential operation within the first year of life". He claims that re-operative cases in adult life are now pursuing their vocations as teachers and even singers whereas before their phonetic ability would not have admitted of any such profession.

MECHANISM OF ASTHMA OF REFLEX ORIGIN. Torrini, of Florence, in the Arch. Ital. di Otol., rinol. e laringol., XXXIX Fasc. 9, 1928, reports that this type of asthma is due to a pressure inequality within the nose. Irregular occluding cells produce uneven pressure thereby allowing intermittent stimuli at different points within the nose which give rise to reflex asthmatic attacks. He has shown that if the nose be tightly packed so that an even pressure is exerted at every point, no reflex attacks will take place.

FISHER, of London, in The Journal of Infectious Diseases, January, 1929, reports a bacteriological study of 46 middle ear infections. From 15 of his cases he intranasally inoculated the pus on the mucous membrane of 15 rabbits after swabbing the rabbits' mucous membranes with 50 per cent phenol. Eleven of these rabbits died with acute purulent rhinitis and acute otitis media. In some of these cases he found as associated reasons marked pleuritis, empyema, lung abscess, acute pericarditis and septicemia. He divided his controls into two groups; one group received only the phenol on the mucous membrane and the other group receiving only the cultures. None of these rabbits died. He gathers from these results that the phenol lowers the local resistance and allows the culture to act more vigorously. He states that this is evidence that otitis media is not blood-borne but occurs by direct extension from an upper respiratory infection.

CORYLLOS and BIRNBAUM, of New York, in the Archives of Surgery, January, 1929, consider lobar pneumonia as atelectasis and broncho-pneumonia as patchy atelectasis. They state that post-operative massive atelectasis is shown to have the identical pathogenesis and evolution as well as the identical clinical and X-ray signs. Because of our knowledge of bronchoscopic therapy in massive atelectasis they bring up the question as to how much good may be expected from the bronchoscopic treatment of pneumonia. As yet they have too few cases to report.

EAGLETON, of Newark, in the March 2, 1929, J. A. M. A., reports a study of the Ophthalmic Examinations in Suppurative Diseases of the Brain. Despite the fact that there is a footnote stating that the article is abbreviated it still retains that clarity and scientific acumen for which Eagleton is noted. Undoubtedly much more is to be learned of the pathology going on within the brain, and the eyes give us a direct route of study. From the discussion of this paper by Suker, Greenwood, Derby, Sterne and Weeks, it appears that ophthalmologists and neurologists are not in complete agreement with Eagleton. This is not the first time that Dr. Eagleton has had to fight his way through much opposition in his deductions.



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